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THE PATHOLOGICAL PHYSIOLOGY OF SO-CALLED PYRAMIDAL SYNDROMES.

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FOR many years the classical findings in cases of occlusive or apoplectic hemiplegia have been described as consisting of immediate post-catastrophic flaccid paresis, followed after an interval by gradually increasing muscle tone and the ultimate development of spastic hemiplegia. However, cases are not infrequently encountered in which various anomalous signs are present, such as early spasticity, late flaccidity, or a combination of flaccid paresis with exaggerated deep reflexes. In most clinical reports either such findings have been ignored or else little attempt has been made to interpret them in terms of disordered neurophysiology. The present paper represents an attempt to correlate the clinical findings, both usual and anomalous, in cases of disease in which the classical signs of a "pyramidal lesion" may be said to exist, with recent studies on the anatomy and physiology of the cerebral cortex and spinal cord.

Anatomico-Physiological Considerations.

Corticospinal Tracts.

Recent histological and physiological studies have made it necessary to revise the traditional concepts of the structure and function of the so-called pyramidal tracts. Thus, Lassek (1939, 1940, 1941, 1942, 1944, 1945, 1946) has shown that, although there are about 34,000 Betz cells in area 4 of each cerebral hemisphere (a Betz cell being defined as one whose diameter is greater than 900μ), there are many more fibres than this (over one million) in the corticospinal tracts; indeed only 2% of corticospinal fibres (those between 11μ and 20μ in diameter) arise from such cells. In addition, von Bonin (1944) has demonstrated that the Betz cells from which these 2% of fibres arise are located only in the upper region of area 4, which he designates as area 4γ (see Figure I). The Betz cells are found in the fifth layer of this area; smaller pyramidal cells contributing corticospinal fibres occur in other layers within this area and in other regions of the cortex (*quod vide*).

A further 30% of corticospinal fibres (that is, additional to those from Betz cells) are derived from the smaller pyramidal cells in area 4 (Lassek, 1942); but the remaining 68% of the fibres in the corticospinal tracts arise from cells situated in regions of the brain other than the precentral gyrus—a fact which is in accord with recent physiological studies (for example, Dusser de Barenne *et alii*, 1938, 1941; Walshe, 1942, 1943; Hines, 1947), which reveal the wide extent of the electrically excitable motor cortex. Recent studies by Levin and Bradford (1938), by Peele (1942), by Tower (1944) and by Gobbel and Liles (1945) have shown that areas 4, 3, 1, 2, 5 and 7 in the monkey, and possibly area 6 in man, contribute fibres to the corticospinal tracts; but this leaves 50% of pyramidal fibres still unaccounted for (Tower, 1944). These may perhaps arise subcortically, as is suggested, for example, by the recent studies of Woodburne *et alii* (1946).

The site of termination of the pyramidal fibres has been studied histologically by E. C. Hoff and his colleagues (1932, 1934, 1935) and by Minckler (1940, 1941, 1944), while physiological investigations have been carried out by David Lloyd (1941, 1942, 1943, 1944, 1946). These workers have shown that the primary termination of the fibres in the lateral corticospinal bundle is upon internuncial neurons (Figure IV) in the dorsal half of the spinal grey matter (dorsal nucleus of Lloyd). Moreover, a large proportion of the uncrossed fibres in both the lateral and ventral corticospinal bundles terminate upon

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neurons in the intermediate portion of the ipsilateral grey matter (Figure V); probably the majority of these latter fibres are derived from the posterior premotor region in area 6 (Fulton *et alii*, 1934; Hoff, 1935; Kennard, 1935; Lloyd, 1946). Each corticospinal bundle contains fibres of many different diameters, a fact which has physiological implications consequent upon the extensive studies of Gasser (1927, 1934, 1935, 1939) and his colleagues upon the rate of transmission of impulses in relation to fibre diameter.

The majority of corticospinal fibres are of small diameter, five-sixths of them being less than 3μ in diameter (Häggqvist, 1936, 1937), while only 4% exceed 10μ in diameter (Lassek, 1939). Most corticospinal tract fibres (about 89·5%) are between 1μ and 4μ in diameter. Moreover, unmyelinated as well as myelinated fibres are found in the corticospinal tracts, 39% of their constituent fibres possessing no myelin sheath (Lassek and Rasmussen, 1939). These findings imply that each corticospinal bundle contains fibres which are capable of transmitting impulses from a wide area of the cerebral cortex, at different rates, and with different physiological results at the local spinal level.

In all mammals both crossed and uncrossed fibres are found in each lateral corticospinal bundle, the latter group constituting about one-tenth of the total corticospinal projection (Fulton and Sheehan, 1935; Fulton, 1946). The crossed fibres therefore form about 70% to 80% of the total corticospinal projection. In the primates the crossed fibres appear to pass only to the lower segments of the spinal cord (Fulton and Sheehan, 1935). The corticospinal tracts are found to have their maximum development in animals with opposable digits, a fact first pointed out by Spitzka in 1886; there are few corticospinal projections in ungulates and cetaceans. Moreover, the ratio of uncrossed to crossed fibres in the lateral corticospinal bundles is altered in favour of the former group as the phylogenetic scale is ascended, particularly with regard to the upper regions of the cord.

Spinal Grey Matter.

Recent investigations on the spinal cord also necessitate extension of the classical concepts of the functions of the spinal grey matter, for within this grey mass is contained the intercalator system of the spinal reflex mechanism, the activity within which governs the output of the anterior motor neuron pools.

Throughout the phylogenetic history of the primate nervous system the afferent and efferent limbs of the originally simple reflex arcs have retained their fundamental constitution, undergoing relatively little alteration in intrinsic structure. It is in the elaboration of the unit interposed between the afferent and efferent limbs that the progressive increase in complexity of connexions and multiplication of cells which has eventually produced the central nervous system in *Homo sapiens* can be traced.

In primitive animals response to a specific stimulus is more or less limited as regards variety. As the phylogenetic scale is ascended, the possible number of responses to any stimulus increases enormously; and this "increased irritability" is the expression of an increase in the number of cells interposed between the sensory receptor and the motor effector units. The larger the number of synapses through which an impulse has to pass in travelling from receptor to effector, the greater the possibility of modification in the ultimate response, because at each synapse the recipient neuron receives impulses not only from the cell immediately preceding it in the reflex arc, but also from others at the same, at higher and at lower levels. It is by this means that the phenomena of summation and inhibition influence the activity of the final common path to the effector unit.

At first reflexes are intrasegmental. Gradually suprasegmental systems are established as the result of the addition of longer and longer chains of intercalators, until finally the degree of complexity becomes almost infinite, the possible responses to a stimulus become incalculable. The cerebral cortex of man is an intercalator mechanism

FIGURE I.

Lateral aspect of the human cerebral cortex, showing, in fine stipple, the regions from which fibres are contributed to the corticospinal tract. Also shown are the histological subdivisions of area 4 and the probable sites of the four suppressor bands, only one of which (area 4s) has yet been identified in man.

of the utmost complexity, in which the possibilities of response are astronomical.

Within the spinal cord itself the intercalator systems vary in their degree of elaboration. A few of the intraspinal fibres pass directly from posterior to anterior horn in the same segment. Others ascend several or many segments before completing the arc. Still other arcs have interposed between posterior horn cell and anterior horn cell one or more neurons constituting a complex cellulofibrillary mass in the grey matter of the cord. In recent years this internuncial system has been the subject of extensive study.

The functional aspects of this complex internuncial system (Figures IV and V), stretching throughout the neuraxis from the oculomotor nuclei to the caudal end of the spinal cord, have been minutely examined by David Lloyd (1941, 1942, 1944). He has shown that the cellular components of the spinal grey matter have a functional as well as a histological orientation, and that changes in their activity are an essential step in the transmission of impulses to the motor neurons from both pyramidal and extrapyramidal sources. Together with the afferent and efferent limbs, they form complex intraspinal reflex arcs which are in a state of constant activity under normal circumstances. This activity is modified by segmental and suprasegmental sources, to the end that discharge eventually occurs from the related motor neurons when their threshold has been reached as a result of summation at the premotor neuron level.

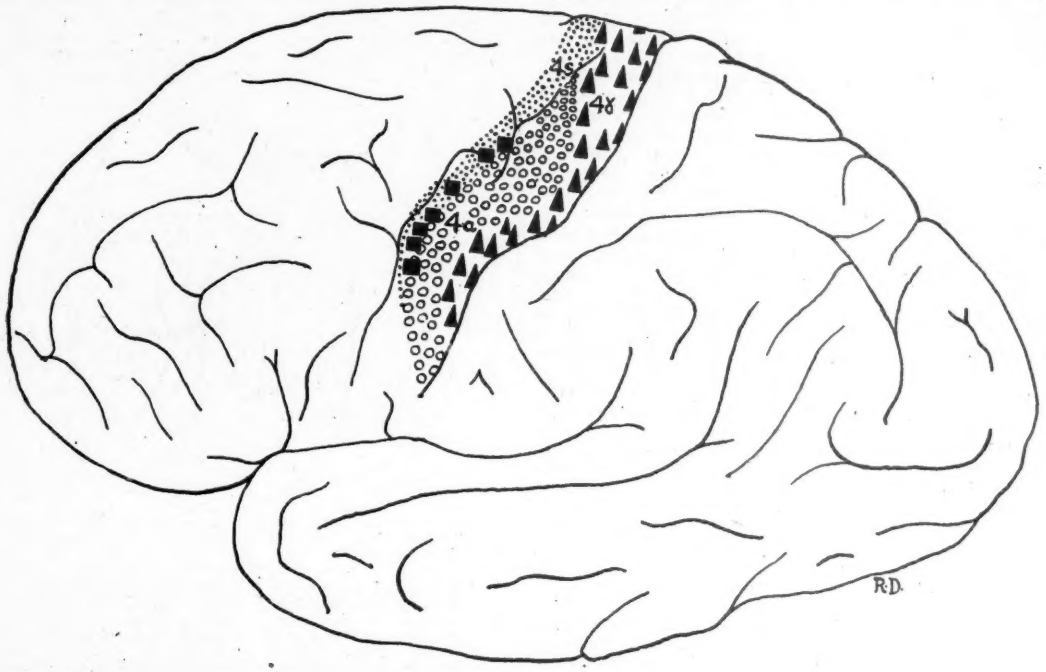

FIGURE II.

Lateral aspect of the human cerebral cortex, showing the subdivisions of the precentral gyrus according to the histological studies of von Bonin (1944). Black squares indicate the suppressor points which have so far been identified in man.

The Posterior Spinal Nuclei.

As a result of Lloyd's studies it is apparent that from the functional point of view the nuclei of the spinal grey matter may be subdivided into three primary groups (Figure IV). Most dorsally situated is a nuclear complex which may be referred to as the dorsal nucleus of Lloyd. This contains two subsidiary cell masses, a medial group, originally identified histologically by Lenhossék (1895), and a lateral group first described by Ramón y Cajal (1909). Between these two groups of cells is a central nucleus, which represents the remainder of the *nucleus proprius* of the dorsal horn originally described by Waldeyer (1891). The dorsal nucleus of Lloyd receives its afferent connexions mainly from the lateral corticospinal tract, and represents the initial internuncial relay in the intraspinal diffusion of impulses of corticospinal origin; other afferents are derived from the local spinal reflex arcs. Schaeffer (1899) and Kappers (1908) had previously suggested, on histological grounds, that pyramidal fibres entered the base of the dorsal horn, terminating in cells situated therein, and this has now been confirmed physiologically. These cells then project to the more anteriorly placed groups of cells in the spinal grey matter. Thus it is apparent that the traditional description of the direct termination of the so-called pyramidal fibres upon cells in the anterior motor neuron pool has little foundation in anatomy or physiology. Impulses of pyramidal origin are filtered through a complex series of intercalator neurons between posterior and anterior grey cornua before their final discharge upon motor neurons.

Anterior to the dorsal nucleus of Lloyd is an intermediate nucleus which consists of two elements, a medially situated Clarke-Stilling column and a laterally situated interstitial nucleus of Cajal. The former nuclear group represents an intermediate relay station on the spinocerebellar pathway. The cells of this system are concerned chiefly in the transmission of impulses related to proprioceptive sensation from the limb musculature and will not be discussed further in this paper.

Cajal's nucleus, on the other hand, is of considerable importance in pyramidal function, because it is the activity of this cell mass that determines the activation of the motor neurons, as is demonstrated by variations in the form of the action potential of the motor neuron discharge in response to bombardment of varying intensity from the intermediate cells (Lloyd, 1941). The total motor neuron discharge is in strict parallelism with the activity

of the intimately related interneuron pools. This intermediate group of cells in turn depends to a considerable extent upon the dorsal group for its activation.

More laterally in this region of the spinal grey matter is situated an intermedio-lateral nucleus whose afferents are derived from the hypothalamicospinal bundle described by Beattie *et alii* (1930) and by Magoun (1940); its efferent fibres constitute the preganglionic fibres of the sympathetic system. Some afferent fibres, however, are derived from the corticospinal bundle either directly or as collaterals, and this may be the basis for some of the autonomic disturbances which are known to occur in cases of subcortical lesions of the corticospinal system (for example, bladder and rectal disturbances, vasomotor changes and alterations in sweat secretion).

Commissural Nuclei.

The commissural nuclei are represented by small congeries of cells located on the medial border of the anteromedian fissure of the cord. They are most numerous in the cervical and lumbar enlargements, at which levels decussating paths are most in evidence (Figure V) and project to the contralateral motor neuron pools. Their afferents are derived mainly from long propriospinal fibres, which, together with fibres of reticular origin, constitute a continuous correlation system extending the length of the neuraxis. Thus the commissural nuclei receive impulses directly from the extrapyramidal projection systems and relay them to contralateral motor neurons.

The Anterior Spinal Nuclei (Motor Neurons).

The motor neurons which innervate the somatic musculature are located in the anterior grey cornua, there being about 1500 to 2000 such cells in the anterior horn of each thoracic segment. They are arranged in two principal subdivisions—a medial group of cells concerned with the innervation of the axial musculature and a lateral group supplying the muscles of the limbs. It is the multiplication and expansion of this latter group in the regions of cord related to the limb girdles that produces the cervical and lumbar enlargements of the anterior grey cornua from which arise the bronchial and lumbosacral plexuses. These cell groups are further subdivided into numerous nuclei, each of which is disposed within certain segmental levels in the spinal cord. In addition to this segmental arrangement there is a longitudinal organization of the nuclei in terms of the individual muscles which they innervate. Representing each of the muscles centrally is a column of cells disposed longitudinally in the anterior grey substance. Each of these vertical columns constitutes the motor neuron pool for the related muscle and its discharge output determines the activity of the particular muscle. Each anterior motor neuron pool represents the final common pathway (Sherrington, 1906) to its related muscle and into it are finally channelled all impulses controlling the tonus of that muscle. Each individual anterior motor neuron likewise represents the final common path to the related group of muscle fibres (usually about 150 in number) innervated by the telendendritic branches of its axon. This latter system constitutes what Sherrington (1926) called the motor unit, a concept which has important implications in the interpretation of primary disorders of the motor neuron (for example, poliomyelitis).

The anterior motor neuron pools depend for their primary activation upon impulses from the interstitial nucleus of Cajal, but in addition they receive afferent impulses from the propriospinal system and the long spinal tracts located in the antero-lateral white funiculus of the cord. These last-mentioned tracts (consisting of the vestibulospinal, tectospinal, reticulospinal and olivospinal tracts) are inhibitory in their effect upon the total motor neuron discharge, with the single exception of the vestibulospinal bundles.

The researches of David Lloyd (1946) have demonstrated that the bulbospinal tracts (see Figure VI) in the antero-lateral funiculus (derived from the tectal, vestibular, inferior olivary and lateral reticular nuclei) constitute an extensive correlation system extending the length of the cord. This bulbospinal system, exclusive of the vestibulospinal tracts, probably represents the descending pathway of the cortical suppressor system described below, and is principally responsible for conveying suppressor activity to the local spinal level. It may thus be referred to as the suppressor bulbospinal system. However, this inhibitory system may itself be inhibited by impulses derived from the vestibular system, the nuclei of which project to the reticular nuclei; so the output of activity at the motor neuron level, from the total bulbospinal system (that is, suppressor system *plus* vestibular system), is the result of the interplay between cortical suppressor and labyrinthine excitor activity at a medullary level.

The fibres constituting the total bulbospinal system are of large diameter and consequently conduct impulses at high velocity. They discharge upon neurons either in the anterior motor neuron pools or upon cells forming the short propriospinal systems of the anterior grey cornua.

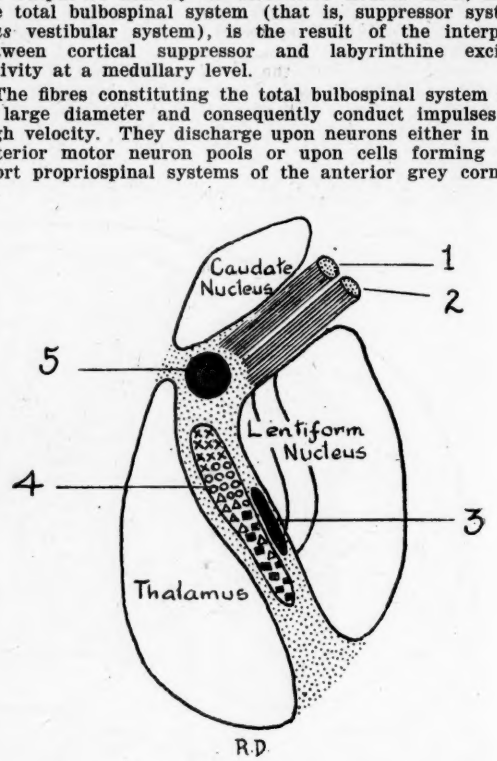

FIGURE III.

Horizontal section through the right internal capsule, showing the relations of the excitatory and suppressor fibres in the corticospinal bundle. Shown in fine stipple are the cut fibres of the thalamic radiations. Squares indicate fibres derived from area 4; triangles, fibres from area 4s; circles, fibres from area 6; and crosses, fibres from area 8. It will be noted that, while there is a definite topographical organization, some intermingling occurs at this level. 1. Frontopontine tract. 2. Anterior thalamic radiation. 3. Corticorubral tract. 4. Corticospinal tract. 5. Corticobulbar tract.

These last-mentioned circuits discharge upon the spinal motor neurons. All this occurs if the bulbospinal system is activated alone and takes place against a background of asynchronous activity in the internuncial systems of the anterior horn (Lloyd, 1946). This latter pathway provides an outflow for impulses from supraspinal levels, which are related both to excitation (vestibulospinal tracts) and inhibition (suppressor bulbospinal system) of motor neuron discharge. It is probably by way of such an efferent pathway (the suppressor bulbospinal outflow) that suppression of muscular response occurs following activation of suppressor circuits in the forebrain.

If pyramidal or local spinal reflex activation sets up internuncial discharge in neurons more dorsally situated in the spinal grey matter, the short propriospinal neurons no longer exert the principal controlling influence upon total motor neuron discharge. This becomes vested in long spinal correlation systems (Figure VI), which then discharge directly upon the motor neurons instead of by way of the short propriospinal system of neurons. Thus

pyramidal or local spinal activity results in a change in extrapyramidal control from a three-neuron to a two-neuron relay system, a concept that has important implications in the interpretation of spinal facilitation and inhibition and of the central excitatory state.

In brief, then, it is apparent that the discharge from the anterior motor neuron pools represents the algebraic summation of excitation derived penultimately from the dorsal spinal grey nuclei and inhibition derived from the suppressor bulbospinal system, the two influences being balanced against a background of tonic excitatory activation derived from the vestibular system.

The mechanism of spinal inhibition has been recently studied by Brooks and Eccles (1947), following their earlier work on the mechanism of synaptic transmission. They have suggested that local spinal inhibition is an electrical effect probably mediated by Golgi II cells located in the anterior motor neuron pools and in the intermediate nuclei. All their findings are in accord with the picture of spinal cord function which has been drawn above, with the modification that inhibition may occur not only within the anterior motor neuron pools but also in Golgi II cells in the interstitial nucleus of Cajal.

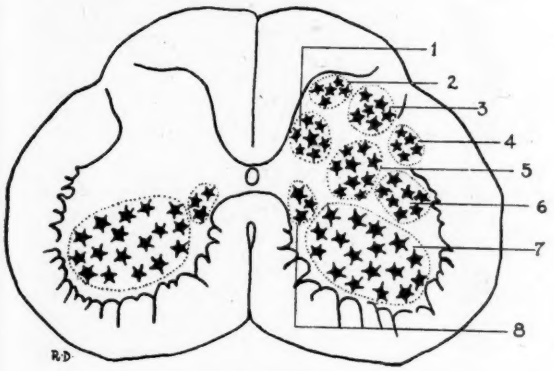

FIGURE IV.

The nuclear organization of the spinal grey matter in terms of the recent studies of Lloyd and others. 1. Clarke-Stilling nucleus. 2. Cells of Lenhossék. 3. *Nucleus proprius* of Waldeyer. 4. External basilar cells of Cajal. 5. Interstitial nucleus of Cajal. 6. Intermedio-lateral nucleus. 7. Anterior motor neuron pool. 8. Commissural nuclei.

Cerebral Cortex.

The above description of the modern view of corticospinal and local spinal function demands also some reference to recent work on the physiology of the sensorimotor cortex. The researches of many workers, too numerous to refer to in detail in this paper, have established a concept of cortical motor function which diverges in many respects from the classical theory of punctate localization. Thus Dusser de Barenne and his colleagues (1933, 1934, 1937, 1938, 1939, 1941, 1942), Hines (1943, 1947), Wagley (1945) and others have shown that the electrically excitable cortex extends far in front of and behind the central sulcus of Rolando and is in part coextensive with those regions of cortex to which the thalamus projects (that is, sensory cortex). One must therefore speak of a sensorimotor cortex and abandon a concept of a "motor" and a "sensory" cortex separated by the central sulcus; differentiation must also be made between "pyramidal" outflow (that is, from Betz cells in area 4γ) and corticospinal outflow (from the electrically excitable regions of cortex). These workers have demonstrated the remarkable lability of the so-called "motor points" in the electrically excitable cortex, in terms of the responses obtained by repetitive stimulation, and they have demonstrated that there are marked variations in threshold of stimulation in various regions of the total excitable cortex. In general, the threshold is lowest in the precentral region and increases with distance anteriorly and posteriorly from this region. This has been discussed in detail by Walshe (1943), who has pointed out that a single stimulation of any point on the cerebral cortex is capable only of sampling the total potential functions of that cortical point. Some of the clinical implications of this have been previously discussed by Wyke (1944).

In addition it has been shown (Hines, 1936, 1937; Dusser de Barenne and McCulloch, 1938; Bailey *et alii*, 1940, 1944; de Barenne and McCulloch, 1941; Hines, 1943; McCulloch, 1944; von Bonin, 1944; McCulloch *et alii*, 1946) that regions may be found within the sensorimotor cortex, activation of which inhibits the muscular response produced by excitation of points within that cortex. Five such regions (Figure I) have been described in each cerebral hemisphere (Verhaart and Kennard, 1940; von Bonin, 1944; Bailey *et alii*, 1944; Glees, 1944; Levin, 1944; Smith, 1945; McCulloch *et alii*, 1946; Magoun, 1946), and these have been called suppressor bands. They are

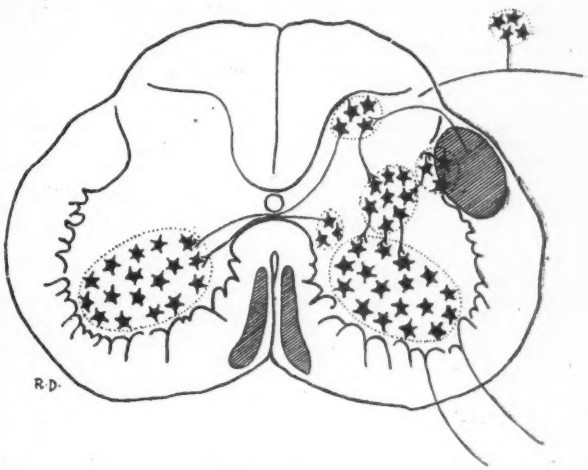

FIGURE V.

Showing the paths of diffusion of pyramidal impulses through the spinal grey matter. Note that both commissural nuclei and the cells of Lenhossék send fibres to the contralateral motor neuron pools.

designated areas 8s (in the frontal lobe), 4s (in the precentral region), 2s (in the postcentral region), 19s (in the occipital lobe), and 24s (in the anterior portion of the *gyrus cinguli*), and they all possess complex connexions with various subcortical and bulbar centres. Only one suppressor band (area 4s—see Figure II) has been so far identified in man, histologically by von Bonin (1944) and physiologically by Wyke (1947). Recent studies by Hines (1947) on the brain of the macaque suggest that the cortical suppressor system may have differential relations to the control of spasticity in flexion as opposed to spasticity in extension. Stimulation of area 4s inhibits contralateral tonic extension, while stimulation of area 6 bilaterally inhibits tonic flexion. These regions are connected, *inter alia*, to the palæocerebellum, the activity of which plays an important part in the regulation of muscle tone both in flexion and in extension (Wyke, 1947).

From these suppressor bands descending fibres pass in a topographically organized fashion through the internal capsule (Figure III) to the caudate nucleus, which then sends relays to various subcortical nuclei (including the red nucleus, *substantia nigra*, *globus pallidus* and subthalamic body) and the thalamus (Papez, 1940; Glees, 1945). Recurrent fibres also send relays back to the cerebral cortex. Within the internal capsule the suppressor fibres are aggregated together in a bundle at the caudal end of the posterior limb, lying mainly behind the other corticospinal fibres but in front of the pyramidal fibres. From the centres mentioned above secondary relays

pass down the brain stem (Glees, 1945) to the inferior olive and thence to the palæocerebellum (Brodal, 1940) and to the reticular nuclei (Levin, 1944). From these nuclei arise most of the descending fibres of the suppressor bulbospinal system previously described. It has been shown (Magoun and Rhines, 1946; McCulloch, Graf and Magoun, 1946) that stimulation of the bulbar reticular nuclei leads to suppression of peripheral muscular activity and that these nuclei are activated by stimulation of area 4s. That stimulation of area 4s in man also inhibits peripheral muscular activity has been shown by Wyke (1947). Further details of this complex subject may be sought in Bucy's book "The Precentral Motor Cortex", published in 1944; but it would seem, on the basis of the available evidence, that the reticulospinal system constitutes an efferent pathway (from higher centres to the local spinal reflex systems) whose activity is expressed as suppression of anterior motor neuron discharge.

A concept of considerable importance to the present discussion is one of a complex series of internuncial relays which are prolonged upwards from the spinal cord into the brain stem. Thus one may picture a series of long and short propriospinal fibres extending by interrupted pathways from the cerebral cortex to the spinal cord. The activity within this system is determined primarily from three sources: (i) the suppressor areas of the cerebral cortex; (ii) the labyrinthine system; (iii) the palæocerebellum. Whether excitation or inhibition occurs at the local spinal level is determined by the relative balance between the corticopalæocerebellar inhibitory system on the one hand and the vestibular excitatory system on the other. Both of these systems discharge onto the reticular nuclei as well as sending more direct fibres down the cord in the antero-lateral funiculi. The long propriospinal correlation fibres which constitute the spinal outflow of this system discharge upon the anterior motor neuron pools and provide a background of balanced activity (both excitatory and inhibitory) against which graded responses of muscle are elicited by corticospinal or reflex spinal excitation of the more dorsally situated internuncial neurons of the spinal grey matter.

Clinical Considerations.

In the light of the foregoing review it seems possible to attempt some interpretation of the varying clinical pictures seen in cases of hemiplegia on a neurophysiological basis. In general, the clinical signs seen in such cases may take one of three forms, which may progress one into the other, but any of which may occur initially at the onset of the symptomatology.

The first clinical picture—and the one most frequently seen immediately following an apoplectic catastrophe—is the well-known state of flaccidity and areflexia in the hemiplegic limbs. This may be explained on the following basis in the case of lesions in the internal capsule. The tonic impulses converging on the nucleus of Cajal (from corticospinal activation of the nucleus of Lloyd) disappear as a result of impairment of function of the corticospinal system in the internal capsule, leaving only the tonic impulses from the vestibulospinal system to maintain subliminal activity in the anterior motor neuron pools. This they have to do against the background of inhibition provided by the suppressor bulbospinal correlation system, the suprabulbar fibres of which tend to escape damage from rupture of a vessel near the caudal end of the posterior limb of the internal capsule (a common site for such a lesion).

It is therefore apparent that the resting activity of the premotor neuron and anterior motor neuron pools, under such circumstances, is below the threshold necessary for continuous discharge of the motor neurons, and hypotonia is the result. Moreover, activation of local spinal reflexes is incapable of elevating this subthreshold activity to the level necessary for discharge, in the face of the inhibitory influence of the suppressor system, and therefore the production of reflex contraction of muscles by stimulation of their stretch receptors is impossible. However, under conditions of maximal vestibulospinal discharge it is theoretically possible to increase the subliminal activity of the anterior motor neuron pools to a point where they may be excited by local spinal reflex activation. This is known to occur under clinical conditions, for it has been demonstrated that in such patients changes in head posture or labyrinthine stimulation by other means (caloric or galvanic stimulation) may be accompanied by alterations in muscle tone, particularly in the extensors of the lower limbs, even though such limbs may be initially in a state of flaccid paresis. Moreover, under such circumstances the stretch reflexes may return, although in abnormal patterns.

A second clinical picture that may be presented, less often initially and more often a few days after the accident, is a state of flaccidity associated with an increase in the deep reflexes. The five cases cited at the end of this report exemplify this condition. This could be due either to

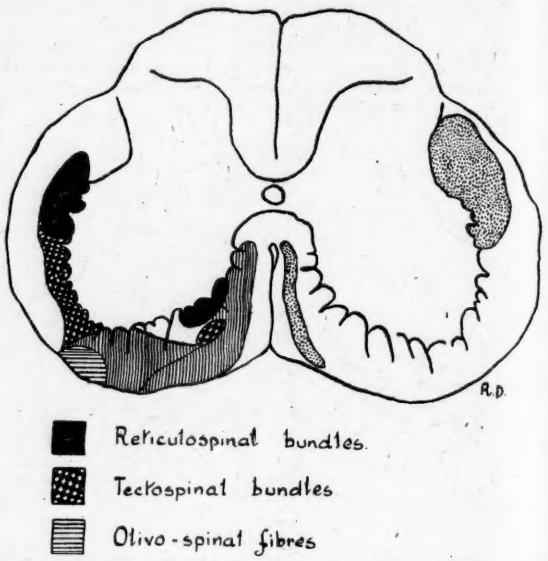

FIGURE VI.

Showing the principal descending excitatory and suppressor fibres in the funiculi of the spinal cord. On the left are seen the fibres of the bulbospinal system, all of which are inhibitory, except those from the vestibular system. On the right is represented the corticospinal excitatory system.

diminished activation of the nucleus of Cajal from corticospinal sources via the nucleus of Lloyd or by increased inhibition of the anterior motor neuron pool from the suppressor bulbospinal system or to a combination of both effects. As a result of the studies of Verhaart and Kennard (1940), of Levin (1944) and of Tower (1944) the latter of these would appear to be the more correct explanation. These workers have shown that the fibres derived from the cortical suppressor bands lie in the internal capsule further from the genu than do the other corticospinal fibres, while the activating fibres from area 4 lie more posteriorly still. The pyramidal fibres related to the upper limb are partially intermingled with the suppressor fibres, while those concerned with muscular activity of the lower limb lie more posteriorly.

Therefore, to account for the occurrence of flaccidity *plus* increase in the local spinal reflexes, it may be postulated that the suppressor bands in the internal capsule either escape more completely or recover more effectively from the lesion than do the corticospinal fibres, and that some residual activation of the anterior horn

cells is going on, derived from remaining homolateral and crossed contralateral corticospinal tracts. The anatomical possibilities of this are evidenced by the fact that the cells of Lanhossék send their axons to the fibres of the opposite anterior motor neuron pool, and also that there are many homolateral fibres in the lateral corticospinal tract (*vide supra*). However, such activation is insufficient to provide adequate discharge to maintain normal muscle tone in the face of inhibition from the bulbospinal system, but is adequate (together with that from the vestibular system) to provide a background of discharge, which is elevated to threshold values by activation of the local stretch reflexes.

The presence of muscular hypotonia also potentiates the reflex responses (when elicitable) as a result of the disappearance of the graded action of the antagonist muscles, consequent upon absence of activation of Cajal's nucleus in the face of persisting inhibition of the motor neuron pools of the antagonistic muscles by the suppressor bulbospinal system. A lowering of the minimal stimulus necessary to elicit the response, and an increase in amplitude of the response together with the appearance of a pendulum jerk, are the result of these effects.

A similar combination of circumstances may also be brought about by a cortical lesion restricted to area 4, or its projections without involvement of area 4s, as has been demonstrated by Hines (1937). This appears to have occurred in one of the cases (Case I) described below. Encroachment upon area 4s results in hypertonia (spasticity), as the inhibitory component is thereby removed and the pendulum effect disappears, although hyperreflexia remains.

The above argument would seem to reduce itself to the fact that diminished activation of anterior motor neuron cells is possible in certain cases to a point such as to cause diminution in muscle tone without, at the same time, impairing the background of discharge beyond the degree necessary for the elicitation of the local spinal stretch reflexes. Moreover, once the stretch reflex is elicited, the response is exaggerated and pendular because of the absence of the normal graded response of the antagonist muscles.

The third type of symptomatic pattern is the one well known by all clinicians to occur in the later stages of catastrophic hemiplegia; this is the stage of hypertonia and hyperreflexia.

In patients manifesting this hypertonic-hyperreflexic syndrome one may postulate that the maximal damage has been towards the anterior end of the posterior limb of the internal capsule (that is, near the genu). Evidence for this is provided by the observation that in such cases the paresis is always more pronounced and recovers less adequately in the arm and face than in the leg. One may suggest, therefore, that the lesion involves the descending suppressor pathway situated posteriorly (but in front of the pyramidal fibres) in the internal capsule, and that the fibres of the excitatory corticospinal system are less severely affected than are those of the suppressor system. In cases of flaccidity *plus* hyperreflexia the lesion is probably more posteriorly situated in the internal capsule (*vide supra*).

In cases of hypertonia and hyperreflexia it would appear that the nucleus of Cajal receives adequate activation from the corticospinal system through the dorsal nucleus of Lloyd, while there is diminution in the discharge from the antero-lateral suppressor bulbospinal system upon the anterior motor neuron pools. Thus the anterior motor neurons continue to be activated from both dorsal spinal and vestibular systems, while the controlling effect of inhibition is diminished.

Conclusions.

It would appear that lesions restricted to the corticospinal projections from area 4 are not the only determining factors in the peripheral clinical picture of hemiplegia. It is well known that lesions localized in this bundle or to area 4 of the cortex produce only flaccid paresis (Tower, 1942), while a lesion extending forwards from area 4 into area 4s results in the onset of spasticity (Hines, 1937). It is not unreasonable to assume that if this is the case at the cortical level similar relations should exist with regard to the projections from these areas. In other words, as has been pointed out above, the discharge from the anterior motor neuron pools (which determines muscle tone) is the algebraic result of activation from the dorsal grey nuclei and from the vestibular system and inhibition from the suppressor projections in the antero-lateral fasciculi.

The amplitude of the local spinal reflex response is governed by the resting subliminal activity of the anterior motor neuron pools, together with the control exerted by the antagonist muscles. Any disturbance in the innervation of the anterior motor neurons will result in disturbance of the reflex response. Thus a decrease in activation will lead to a diminution or disappearance of the reflex response, while a diminution of suppression and/or impairment of the activation of antagonist muscles will result in an exaggerated response to activation of the stretch receptors.

Thus the quality of muscle tone may be divorced from the quality of the reflex response, by virtue of the dichotomy which appears to exist between activator systems on the one hand and suppressor systems on the other at all levels of the central nervous system.

Reports of Cases.

The following five cases illustrate the differentiation that may occur in cases of hemiplegia, between muscle tone on the one hand and reflex response on the other, as a result of interference with the excitatory and suppressor systems in or descending from the cortex. Such cases are probably much more common than a survey of the literature would lead one to expect. Certainly one may recall numerous instances of such cases from one's own experience; but the tendency has often been to ignore such observations in the absence of an adequate explanation thereof. It is felt that the preceding discussion provides a possible explanation, on the basis of known clinical and experimental data, for the clinical phenomena about to be described. It is considered that the first patient sustained a cerebral thrombosis in one of the vessels supplying area 4 of the cortex, while the damage in the second case was the result of a hæmorrhage into a neoplasm extending subcortically from the left temporoparietal region.

CASE I.—The patient, L.B., a railway worker, aged fifty-six years, was admitted to the Royal Prince Alfred Hospital under the care of Dr. C. G. McDonald on May 2, 1947. Since November, 1946, the patient had suffered from headaches, which had become progressively worse. In May, 1947, the patient had a sudden exacerbation of his headache associated with a sharp pain in his right leg. This disappeared for about ten minutes, and was followed by a second similar attack in which he fell over unconscious. When found, he had regained consciousness, but was unable to move the limbs on the right side of his body or to talk. He was incontinent of urine at the time of the attack and for some time afterwards. He was then admitted to hospital, where he was found to have right-sided hemiplegia, together with urinary incontinence and inability to speak.

On examination, the patient was seen to be a somewhat flushed man whose right arm and leg were motionless. The right shoulder was drooping. He exhibited excessive emotional lability, and a slight right facial paresis was present. The right pupil was larger than the left. There was no impairment of consciousness and the patient understood fully all that was said to him; but he was unable to make coherent replies to questions. Attempts to speak resulted only in the repetition of the word "good" accompanied by much gesticulation with the left hand and by emotional distress. A nominal aphasia was present. The patient was right-handed. Examination of systems other than the nervous system disclosed no abnormality; the blood pressure was 120 millimetres of mercury, maximum, and 90 millimetres, minimum, and the blood count was within normal limits. There was no enlargement of the heart and the pulse showed no abnormality.

Neurological examination revealed no disturbance of olfactory sensation, or of the function of the second cranial nerve. Ophthalmoscopic examination disclosed no abnor-

mality. The right pupil was larger than the left, but all the pupillary reflexes were normal. The fourth and sixth cranial nerves were normal. There was some weakness of the right masseter and the right pterygoid muscles. The sensory component of the fifth nerve was intact. There was a right upper motor neuron lesion of the facial nerve. Bilateral deafness was present, but no abnormality could be found by the Rinné and Weber tests. There were no abnormalities of the other cranial nerves except for some weakness of the right trapezius muscle. There were no disturbances of autonomic function, except for episodic urinary incontinence.

No coordination tests could be performed on the right side of the body. On the left side such tests gave normal results.

Impairment of all modalities of sensation was present on the right side of the body.

There was flaccid paresis of the right arm and leg, more pronounced in the arm than in the leg. Muscular power and tone were normal on the left side. A Babinski response without toe fanning was elicited on the right side; ankle and patellar clonuses were present on the right, and the right knee jerk was exaggerated, the minimum stimulus necessary to elicit a response being less than normal. The right abdominal reflexes were absent, and all the deep reflexes in the right upper limb were augmented. A pendular element was present in the right knee jerk and right triceps jerk. There was some wasting of the right leg and the right arm.

CASE II.—Mrs. B.M., aged fifty-eight years, was admitted to the Royal Prince Alfred Hospital on September 15, 1947. As the patient was drowsy and incapable of adequate cooperation the history was obtained from the relatives. They stated that she had been well until four months prior to her admission to hospital, when she had developed an upper respiratory infection, which was followed by nasal obstruction and cough lasting for two months. At the same time she had several attacks of nausea and vomiting, usually in the mornings. Two months prior to her admission to hospital she began to complain of severe boring headaches, at first most severe in the frontal region, but later spreading round to the occiput and down the back of the neck. With the onset of these headaches the nausea and vomiting became worse and occurred at any time of the day. About two weeks prior to her admission to hospital the patient had a peculiar "turn", during which she felt giddy and experienced a sensation of objective rotation in an anti-clockwise direction. She did not lose consciousness, but was not able to stand. She was put to bed and was prevented from rising by the recurrence of giddiness. Shortly afterwards she lost the use of her right arm and leg, and sensation disappeared in the right half of her body; but both of these effects had waned in the few days prior to her admission to hospital. At the same time she had had incontinence of urine and fæces, but this had diminished lately. During the two weeks before her admission she had had olfactory hallucinations, which preceded exacerbations of her headache. There were no other relevant details in the history except for the occurrence of some mild dyspnœa and palpitation on exertion, with occasional swelling of the ankles.

General physical examination disclosed some slight thickening of the vessel walls and a blood pressure of 165 millimetres of mercury, maximum, and 100 millimetres, minimum. There was slight œdema of both ankles and the heart was slightly enlarged to the left. The other systems (apart from the nervous system) were normal, except that a cervical erosion was present on the posterior aspect of the cervix with contact bleeding and an offensive vaginal discharge. There was no evidence of malignant disease in the pelvis.

Examination of the nervous system disclosed that the patient was somewhat drowsy and non-cooperative. The first and second cranial nerves were normal. The other cranial nerves were also normal, except for slight hypoglossal paresis on the right side. There was no obvious paresis, nor was there any alteration in peripheral muscle tone. Sensation was unimpaired and the reflexes were normal on this occasion. Lumbar puncture revealed slightly blood-stained cerebro-spinal fluid. The supernatant fluid after centrifugation was xanthochromic. The cerebro-spinal fluid pressure was 110 millimetres of water.

On the following day paresis of the right arm was apparent, with extreme flaccidity of the whole limb. On September 22 a second lumbar puncture disclosed xanthochromic fluid at normal pressure. The fundi were normal.

On September 30 the patient had a seizure in the ward and was unconscious for ten minutes. After the seizure she complained of severe headache and of pain in the right arm. Both the legs were hypotonic, with ankle clonus and exaggerated deep reflexes.

On October 1 gynæcological examination disclosed no evidence of malignant disease in the pelvis, and there was no radiographic evidence of metastases in the lungs.

On October 8 headache was severe and neck rigidity was present. Lumbar puncture disclosed blood-stained cerebro-spinal fluid at normal pressure. It contained 100 milligrammes of protein *per centum*. The right arm was still flaccid, but the tendon reflexes in this limb were remarkably exaggerated, with a pendular triceps jerk.

On October 15 the patient was able to move the fingers of her hand, and there was some increase in tone in the right arm. The tendon reflexes were still exaggerated.

Radiographic examination of the skull revealed no abnormalities. Electroencephalography disclosed an unstable α rhythm disturbed by random slow waves, particularly in the left postmotor region. No phase reversals were seen.

The visual fields were normal. The blood and cerebro-spinal fluid failed to react to the Wassermann and Kline tests. The cerebro-spinal fluid Lange curve read "0011110000". The blood count was within normal limits.

CASE III.—L.W., a woman, aged sixty years, was in her usual health until February 15, 1948, when she suddenly noticed that she could not use her right arm and fingers and that she could not articulate clearly. She was admitted to the Royal Prince Alfred Hospital on the same day. On examination she was found to have incomplete right-sided hemiplegia. Dysarthria was present, but there was no loss of the higher speech functions—the patient could understand the written or spoken word, and could name objects. Slight movement was present in the right arm, but there was an absence of movement in the right forearm, and only slight movement was present in the right lower limb. The lower part of the right half of the face was immobile and the tongue deviated to the right. Babinski's sign was present on the right side.

The deep reflexes on the unparalysed side were approximately normal (the biceps jerk may have been slightly increased). On the affected side, however, the deep reflexes—especially the biceps jerk—were much exaggerated, although hypotonus was present. The blood pressure was 190 millimetres of mercury, maximum, and 76 millimetres, minimum. The area of deep cardiac dulness was increased in diameter, and a mitral diastolic and an aortic systolic murmur were present. No information of importance could be elicited with regard to the patient's previous health and family history.

On February 28 lumbar puncture was performed. The pressure of the cerebro-spinal fluid was 190 millimetres of water; the Queckenstedt test produced a positive result and the cerebro-spinal fluid was clear and colourless. Chemical and microscopic examination of the cerebro-spinal fluid revealed no abnormalities. The blood reacted to the Wassermann test. X-ray examination showed that the heart was enlarged in its transverse diameter and atheromatous plaques were present on the aortic knob.

CASE IV.—A.K., a woman, aged sixty-eight years, had suffered from asthma for some years, but had been in her usual health until December 1, 1947, when she collapsed in the street and was taken to the Royal Prince Alfred Hospital. At the time of her admission to hospital she was not unconscious, and although she was unable to reply to questions, she kept up a moaning conversation and was capable of cooperating to a limited extent. On examination she was found to be suffering from left-sided hemiplegia involving the lower part of the face, the tongue, the left arm and the left leg. Both eyes were deviated to the right and the head likewise persistently turned to the right. Hemianæsthesia was also present on the left side. The admitting officer reported that at this time occasional movements were occurring at the left shoulder, and that there was an increase of tone in the left arm and leg. Babinski's sign was present on the left side. The deep reflexes on the right side were normal; on the left side the knee jerk was present, but the ankle, biceps and triceps jerks were absent. The blood pressure was 210 millimetres of mercury, maximum, and 140 millimetres, minimum.

Examination of the ocular fundus showed arteriosclerotic thickening of the vessels. On December 2 lumbar puncture was performed. The cerebro-spinal fluid pressure was 160 millimetres of water and the response to the Queckenstedt test was positive. The fluid was blood stained and on centrifugation was xanthochromic. Neither the cerebro-spinal fluid nor the blood reacted to the Wassermann test. Further examination of the cerebro-spinal fluid on December 9 again showed the presence of blood-stained fluid. On

December 3—that is to say, three days after the onset of the stroke—the deep reflexes of the left arm were found to be slightly exaggerated, although atonia was present. In the left leg the deep reflexes had returned, but were not increased, and tonus of the limb was diminished.

Up to the eighteenth day the deep reflexes of the left arm were elicited with increasing ease, although the complete atonia continued and there was an entire absense of voluntary movement. Meanwhile some voluntary power had returned in the left leg, and although tonus in this limb was less than on the unparalysed side, the knee jerk was elicited with slightly greater ease than on the opposite side.

Meanwhile the hemianæsthesia and conjugate deviation had disappeared.

CASE V.—E.D., a woman, aged eighty-three years, had been in her usual health until February 27, 1948, when she experienced some giddiness while at breakfast and soon afterwards had difficulty in speaking. Several hours later she complained of weakness in the right arm and leg and of increasing drowsiness. Next day (February 28) she was admitted to the Royal Prince Alfred Hospital in a stuporose condition.

On examination of the patient it was found that the right arm and leg were paralysed, but the cranial nerves appeared to be unaffected. Her blood pressure was 200 millimetres of mercury, maximum, and 100 millimetres, minimum. The patient had not previously suffered from any symptoms referable to hypertension, and nothing of any significance could be elicited regarding her previous health, family history or habits and surroundings. When she was examined on March 2 it was found that the right arm was paralysed and atonic and that the deep reflexes—especially the biceps jerk—were increased. The right leg was likewise paralysed and some tonus was present, although this was not so pronounced as in the opposite extremity. The deep reflexes in the leg were also elicited with slightly greater ease than on the left side, but they were not increased to nearly the same degree as in the right arm. The left arm was normal as regards voluntary motor power, tonus and tendon jerks. Babinski's sign was present on the right side.

On March 5 the right arm was still completely paralysed and atonic, while the deep reflexes were more increased than before. In the right leg tonus—although less than normal—had slightly increased, but it was not possible to elicit the deep reflexes on this occasion. Babinski's sign was still present. The left arm now showed an increase in tonus above the normal and the deep reflexes were slightly increased. The left leg was normal as regards voluntary motor power, tonus and deep reflexes. The lower part of the right side of the face now appeared to be slightly weaker than the left side.

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NASAL DESENSITIZATION IN ALLERGY.

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THIS article describes a new method of treating allergic manifestations affecting the naso-respiratory tract. It has been developed as a sequence to the intradermal method of administering allergic vaccines.

The method described in this article has been employed by me for ten years. It is now considered that a sufficient length of time has elapsed to submit a description of the technique used, together with an opinion on its merits. It has been used in the treatment of all the usual manifestations of allergy affecting the naso-respiratory system.

Rationale of the Method.

The rationale of the method is based on the production of a local immunity in an area of skin constantly subjected to intradermal injections of vaccine. Whether a true local immunity is produced or the constant injections produce a local toughness or induration of that particular area of skin surface, accompanied by a change in the lymphatic vessels hindering absorption, is not under discussion in this article. If the injections are confined to one particular skin area for a period of weeks and then changed to another area, the patient may experience a definite local or general reaction, or both, even if the dose employed has been the same or smaller than previous doses.

It was thought that if allergic vaccines were injected into the nasal mucosa the resulting general immunity would be at least equal to that obtained by the intradermal method, and in addition the local immunity would also be of value to the patient. In other words, the local immunity would be in the most desirable area. The method is equally applicable to patients giving either strong reactions or weak reactions when the skin has been tested in the course of a routine examination. It is not to be regarded as a complete substitute for the intradermal and subcutaneous methods. Anyone experienced in allergy will know that these two methods have a very well-recognized place in the treatment of patients with allergy. It is, however, to be stressed that the intranasal will do very much more than the other methods. It has been found possible to change from the intranasal to the intradermal method when considered necessary, and certainly to switch from the intradermal to the intranasal. The latter is the usual procedure. For the reason that the skin reactions are not an infallible guide to the manner and type of reaction following an injection of an allergic vaccine, the practice has grown with me of beginning the treatment of a patient with the use of the intradermal method and then of changing over to the intranasal method.

Outline of Response to the Method.

Observation over a period of years has shown that certain varieties of allergy respond more than others to the intranasal technique.

The following is a brief outline of the response to this treatment.

Hay Fever.

There is no doubt that the ordinary uncomplicated condition of hay fever responds better to this method than to the usual subcutaneous technique and even to the intra-

dermal method. Further, a patient who has not responded satisfactorily to the intradermal method will in most cases react very favourably when the technique is altered and this method employed. Patients under treatment are very definite on this point.

Asthma.

Patients suffering from asthma and giving good to moderate reactions to skin tests will react favourably to this technique, in a similar way to those with hay fever.

Allergic Rhinitis.

The group of allergic rhinitis comprises true allergies, borderline allergies, cases of infective rhinitis with a doubtful allergic tie-up, and conditions which do not fit satisfactorily into any of these classes. Patients with these conditions respond in a most satisfactory manner to this line of treatment.

Nasal Polyposis.

Patients with nasal polyposis are the least satisfactory group to deal with, as there are many subgroups. Members of certain of these subgroups have been benefited considerably.

Technique.

Anæsthesia.

An intramucosal injection can be made in the vast majority of cases with a minimum of pain—certainly with less pain than a subcutaneous or intradermal injection. Anæsthesia when employed has the double effect of anæsthetizing and shrinking the nasal mucosa. This enables the operator to work in comfort and minimizes the probable occurrence of reactions.

To secure anæsthesia sufficient for the purpose, the procedure adopted is to place in the nasal cavity over the area to be treated a pledget of cotton wool soaked in a suitable solution. Such a solution is a 1% solution of "Neo-Synephrine", "Tuamine Sulphate" or "Privine" with sufficient cocaine hydrochloride to make a 2·5% solution. A suspension of "Benadryl" 10% in normal saline solution will act as an anæsthetizing solution. The pledget is applied as before. Adrenaline has been discarded, as it has been found unsuitable for the purpose. After adrenaline has been used many patients suffer for hours from uncontrollable sneezing. This disability is unfortunate and peculiar to adrenaline.

Intranasal Injection.

The instruments necessary are the usual nasal gear: frontal mirror, nasal speculum, sufficient illumination, swabs and applicators, one cubic centimetre vaccine syringe and a special nasal needle. The needle consists of an ordinary record or BD socket in which has been fitted a three-inch needle of 25 gauge. The needle is curved sharply at the tip. The needle opening has a short bevel. The needle must always be kept very sharp. If not, the operator will be unable to make a satisfactory injection.

It is generally found convenient to begin on the nasal septum. As the doses increase more of the mucosa is used and more injections are required. It is not always possible, as with intradermal injection, to spread the injection and so produce a large wheal or swelling. Any undue force used will have the effect of rupturing the mucosa and the fluid to be injected will be emitted in a fine spray into the nasal cavity. As the area to be treated extends posteriorly and cranially, more care has to be taken in placing the pledget of wool in the most suitable position.

The right and left nasal cavities are used alternatively. The injection is made into the submucosal layer or into the mucosa itself. It is not possible with some patients to place the injection into the mucosa, as the slightest pressure of the operator on the piston of the syringe causes free leaking and so loss of the injected fluid. When this is noted the injection is made into the submucosa.

The injection of the nasal septum and inferior turbinate bone is comparatively easy. In the case of the inferior turbinate bone the injection is begun at the anterior end and then worked posteriorly. With a hypertrophied anterior end of the inferior turbinate it can be very difficult to inject over the hump onto the posterior portion.

In the case of the middle turbinate it is essential to use a special needle with a sharp, almost right-angled turn about a half-centimetre from the end, care being taken that the bevel of the needle is very short. When the mucosa of the middle turbinate is boggy, this procedure is not difficult, but as one works in a cranial direction it is more and more difficult. It is very necessary to place the anæsthetic pledget of wool correctly to ensure proper anæsthesia. The mucosa over the middle turbinate is always loose and easily movable on the underlying bone; nevertheless it is not easy to place this injection correctly.

It is a matter for experience to know just how much pressure can be used in making these injections. Generally a small puncture hæmorrhage follows the injection. This is easily controlled by a small plug of cotton wool.

Interval between Doses.

When the operation desired is quick desensitization, the interval between doses can be from three to seven days. For general work in the early stages of the treatment weekly injections are desirable.

Reactions.

The reactions following this procedure do not vary a great deal from what one experiences in routine allergic desensitization procedures.

Main Types of Reaction.

Routine Reaction.—The routine reaction varies from mild to severe. It occurs from ten to thirty minutes after the injection is completed. The main symptoms are sneezing, stuffiness in the nose and running eyes. In short, most of the symptoms which accompany a mild attack of hay fever are noted. Only experience can keep reactions in this class. This is the reaction generally observed and the one to be aimed at.

Moderate to Severe Reaction.—A moderate to severe reaction occurs within ten minutes when a pollen or inhalant vaccine is used. The symptoms and signs are the same as in the previous class, but facial swelling is seen and asthma may be produced.

Moderate to Severe Delayed Reaction.—A moderate to severe delayed reaction occurs several hours after the injection is given. It is more or less similar to that described in the preceding paragraph, but there is generally swelling of the face, especially of the circumorbital areas, the lips and the skin of the neck. Urticarial rashes may be scattered over the body.

Protein Shock Reaction.—Protein shock reaction occurs only when bacterial vaccines are used. It takes place several hours after the injection. Violent shivers, headaches and abdominal pains characterize this type. It may be compared with an attack of malaria.

Severe Asthmatic Reaction.—An injection of histamine intranasally into some patients with nasal polypi will produce severe asthma. This is a special class of patient, as all polypi are definitely not histamine-sensitive.

Severe Allergic Reaction.—Severe allergic reaction has occurred twice in a period of ten years and in form does not vary from the response elicited by the subcutaneous method. There are two grades of severity, severe and very severe. A description of each will suffice.

In a severe reaction, within thirty seconds of the injection the patient complains of a burning feeling all over the body. Within a few minutes gross œdema of the face, lips and tongue is seen. A severe asthmatic spasm follows and the patient is soon in a desperate condition.

The second variety was seen when, on one occasion, within three minutes of the intranasal injection, the patient complained of a severe burning pain in the temporal region of the head. This was quickly followed by unconsciousness associated with cyanosis, moderate rigidity and stertorous breathing. Under treatment consciousness returned and the whole condition subsided. Within twenty-four hours the patient was normal again. No adrenaline was given to this patient. No swelling of the face or body was noted and no wheezing in the chest. This patient had had previous injections in the nasal mucosa with the

same vaccine used on this occasion without any untoward reaction. The patient has had no trace of hay fever since.

Further Notes on Reactions.

Routine Signs following the Correct Injection.—The injection is followed by swelling of the nasal mucosa and consequent blocking of the nose. Sneezing is an accompaniment, but is much less frequent than one would imagine. The avoidance of adrenaline in the anæsthetizing solution has contributed a great deal to this end. The nasal stuffiness may be present for several hours after the injection, but is usually completely gone in twenty-four hours. Facial swelling in the region of the inner canthus of the eye may appear on the day of the injection or on the day following and subsides in two or three days.

The Shock Reaction.—The shock reaction is not produced by either inhalant or pollen vaccines. It is not produced by all bacterial vaccines and may not be produced by autogenous vaccines. Its production apparently depends on the strength of the vaccine. Using a strength of one thousand million organisms to one millilitre of solution, one does not experience it with small doses such as 0·025 millilitre. When the operator uses vaccines in strengths of two thousand five hundred million and upwards, reactions are regularly produced. These are very severe and the operator should proceed cautiously before increasing the dosage.

Histamine Reaction.—In the production of reactions histamine solutions will initiate a very severe asthmatic reaction when injected intranasally in certain cases of asthma associated with nasal polypi. The reaction is one which occurs in about twenty minutes from the time of injection and is asthmatic in type. It varies considerably in that it may or may not be produced by the treatment. It has to do with the dose employed, as when one tries to increase the dosage it is encountered. It is the only solution that I have found to reproduce an asthmatic attack in patients suffering from nasal polyposis. I have so far been unable to establish any tolerance for this injection.

Control of Reactions.

It is better not to have severe reactions, and the more experienced the operator, the fewer he will have.

Reactions following intranasal injections can be controlled in the following way. The insertion of a pledget of wool soaked in adrenaline (1 in 1000 solution) into the nostril which has been the subject of treatment will generally control any reaction. Should the operator desire, he can insert an adrenaline pledget into the other nostril or give a subcutaneous injection of adrenaline hydrochloride (1 in 1000 solution), using the dose that he thinks necessary. Generally five to fifteen minims will control any reaction.

Dosage.

There is very little difference in the dosage scale between the intranasal and intradermal methods. The interval between injections is generally seven days, but can be modified to suit the convenience of either the patient or physician. At least four days' interval should be allowed for the nasal mucosa to subside.

When a patient has given strong reactions to testing, the use of the intranasal method without any preliminary testing out of his skin reactivity is only asking for trouble. For this reason it has been my custom to commence the treatment of all patients with intradermal injections at weekly intervals. Having satisfied myself as to their activity and also produced a certain amount of immunity, I proceed with intranasal desensitization. The initial dose is 0·025 millilitre and this dose is persisted with if any reaction occurs and is increased in the absence of a reaction. The usual increase is 0·025 millilitre, and when by gradual increases the dose of 0·20 millilitre is reached, it is found that the scale can be augmented by 0·05 millilitre at each injection. It is usual to inject right and left sides alternately. When the scale has reached one millilitre it is the usual practice to stay at that level for three injections and then to cease treatment for about three months.

Areas of Nasal Mucosa Treated.

The initial doses are placed in the mucosa overlying the nasal septum. The injection is kept in this area, different areas being used at each treatment till the physician is confident that most of the mucosa has been treated. The area next treated is the inferior turbinate bone, and finally the middle turbinate. This bone is left till last, as it is without doubt the most difficult one to treat by this method. By this time the operator will be more skilful, and finally the shrinking of the mucosa that does occur in this procedure renders the actual injection a simpler performance.

The middle turbinate bone is always injected at the caudal or inferior surface and the injection gradually worked along the surface. This bone is always more sensitive than the remainder of the nose and care must be taken with the production of anæsthesia.

Results.

This method has now been practised by me for ten years. No results have previously been published and, as far as I am able to ascertain, the method is not in use elsewhere.

It is my opinion that in some varieties of allergy the method is very much more efficacious than any other.

With pollen hay fever and asthma the method will produce an immunity more quickly than the intradermal method. When intradermal immunization is apparently failing and the results are lagging, intranasal injection definitely helps.

In the case of allergic asthma the results are better than with other methods. The nasal symptoms are lessened, any tendency to hay fever is mitigated, and the increased nasal airway plays an important part in producing a favourable result.

On several occasions patients have affirmed that their nasal symptoms have been relieved out of proportion to their chest symptoms.

That group of conditions classified as nasal allergy, allergic rhinitis, paroxysmal rhinorrhœa and other names is the group *par excellence* that is relieved most by this form of treatment. This is the group most benefited by the local application of "Benadryl".

One of the striking changes indicating improvement is change in the colour of the mucosa from its allergic colour to pale pink.

To test the local effect of the injections on several patients the treatment was confined to the one side. Invariably the patient would remark on the improvement in the airway compared with the untreated side.

The gradual shrinking of the mucosa, *plus* a change in the colour, leads to an increased airway and so to great general improvement. Response is prompt and satisfactory.

In the case of nasal polyposis results of this treatment vary with the particular type of polypus. At present the following has been noted.

1. A patient with nasal polypi who has been treated by intranasal injection has not to date developed asthma as a result of the treatment.

2. Asthmatic attacks can be induced by the injection of histamine in some subjects with nasal polypi and a previous history of asthma. Should it be necessary to remove the polypi, operation can be performed, and so far no case has occurred in which the removal has precipitated the patient into an asthmatic seizure.

3. The results appear more quickly than those from intradermal treatment. The quick disappearance of nasal stuffiness is one of the pleasing features of the treatment.

4. A high immunity is developed against the common cold. More than once patients have expressed themselves as amazed and delighted at their freedom from this disease.

Advantages.

The advantages can be summarized as follows.

1. The patient has no sore arm to hinder him in his daily work.

2. Very little discomfort is produced. As one's skill increases and one's knowledge of how much to give widens, reactions become less and less frequent.

3. Patients seem to be happier when the treatment is directed to the nose and not the skin. It applies the treatment closer to the site of the disease.

4. In ten years there has been reason for alarm in only two cases, although hundreds of patients have been treated and thousands of nasal injections have been given. This record compares more than favourably with any other form of desensitization.

5. The period of time for treatment in my hands has been considerably shortened. The cost of treatment, the time necessary to complete the injection and the economical balance are very much in favour of this treatment.

6. Both local and general immunity is produced. In addition, the local immunity is situated where it is of most value, that is, where the symptoms are produced.

7. Weaker solutions of vaccine can be used and the results obtained so far are better than those from stronger solutions given intradermally.

Disadvantages.

The disadvantages are as follows.

1. It is time-consuming compared with a simple injection.
2. It requires special needles and a certain amount of skill, which can, however, be developed.
3. It is not trouble-free, but neither is any other method.

Conclusion.

Finally, in the vast majority of cases this method is definitely to be preferred, as it does produce results more quickly than any other method. It is not suitable for nor wise to use on every patient. Experience will tell the operator when to use it.

These notes are submitted to offer to practitioners interested in this work a method which will definitely improve their results in the vast majority of cases.

It is hoped to publish at an early date further notes giving greater detail and amplifying the results obtained in a series of patients from the various allergic groups.

PULMONARY TUBERCULOSIS IN SOUTH AUSTRALIA.

By R. B. LEWIS, B.Sc.,
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MUCH WORK has been done, for example by Dr. J. H. L. Cumpston[1] and Dr. M. J. Holmes,[2] in analysing the course of tuberculosis, including pulmonary and other forms, in Australia and the individual States. This present article does not attempt to cover again that ground which has been covered so well.

Instead, the following graphs show the results of a statistical approach to the study of pulmonary tuberculosis in South Australia which is thought to be new, in this State at least. The death rate has been considered not on its own merits, but in comparison with the morbidity rate as shown by the "number of cases of pulmonary tuberculosis notified per 100,000 population". The figures will show that of all cases notified in South Australia, 60% to 70% result in death, and that this percentage is no better—is if anything a trifle higher—than it was thirty-five years ago.

Death Rate and Morbidity Rate.

It will be seen from Figure I that the death rate from pulmonary tuberculosis in South Australia shows a very satisfying decrease throughout this century. The actual number of deaths *per annum* has fallen nearly to half the 1900 figure, and at the same time the population has nearly doubled, so that the death rate is now only approximately one-third of the 1900 death rate.

At the same time, Figure I shows that the number of people being infected by pulmonary tuberculosis, as shown by the notifications of the disease *per annum*, has dropped considerably since about 1910. As notification was introduced by law to South Australia only in 1899, it is possible that the rise in the notification rate from 1899 to about 1910 is at least partly due to the lag in the system of notification being overtaken in these early years.

The two curves on Figure I show considerable fluctuation from year to year. Figure II shows an attempt to smooth out the death and morbidity rate curves by means of taking five-year moving averages of each set of figures.

From this graph it appears that from a peak in 1908 the death rate has fallen remarkably steadily to the

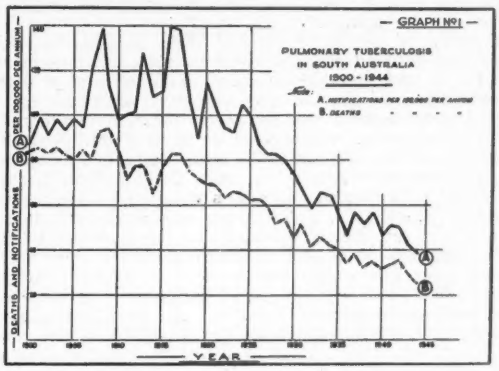

FIGURE I.

present day, with the exception of a peak occurring in 1917, possibly due to war-time influences.[2] In the same period the morbidity rate has fallen from a peak in 1915, again with remarkable steadiness, especially from 1922 to 1944, and even more steeply than the death rate curve.

Comparison of Death Rate with Morbidity Rate.

It is with the relationship between these two curves that the present article is most concerned, and a very simple attempt has been made to compare the two by taking the (averaged) deaths in any one year as a per-

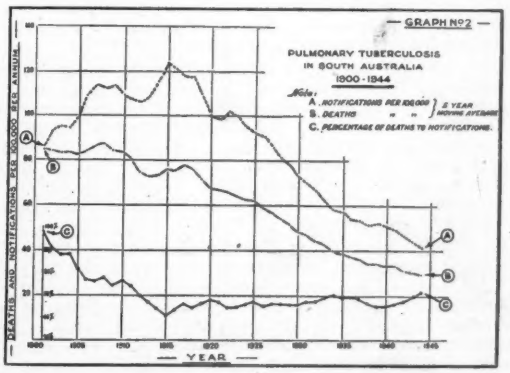

FIGURE II.

centage of the (averaged) notifications in the same year. These percentages are shown in Figure II as a curve, which falls sharply from the 1900 level of nearly 100% to just over 60% in 1915, thereafter maintaining a very steady level until 1944.

This simple method of taking deaths *per annum* as a percentage of cases notified in the same year is open to some criticism, particularly on the grounds that by no means every death occurs in the same year that the patient's case is notified. In defence of the method,

looking again at Figure I, it will be seen that the maxima and minima of the two curves coincide very closely indeed on the time scale. In other words, a year of relatively high notification rate appears almost invariably as a year of relatively high death rate, and similarly for relatively low rates.

Again from Figure II the coincidence of maxima and minima on the two curves is very close. Possibly if the notification curve was moved bodily a year or two to the right,[3] the coincidence would be slightly improved and the percentage curve would then be a trifle smoother and at a very slightly lower level; but its general shape would remain the same, and for a first approximation, therefore, the curve as it stands will be taken.

At this point it might be wise to mention other possible errors in the figures.

Firstly, as Dr. F. S. Hone[4] has shown, the official figures of notified cases *per annum* may not be a correct record of the number of new cases of active disease developing each year in a community. Also the criteria for notification by a medical practitioner involve considerable uncertainty. (The various notification procedures, and varying dates when notification was made compulsory in the several States of the Commonwealth, make Australia-wide figures for years prior to 1929 difficult to obtain. Since 1929, according to the data available, the figure for the Commonwealth has been between about 70% and 85% of deaths to cases notified.) However, it would seem fairly safe to assume that certainly no less, and probably relatively more, of the cases of active disease are notified nowadays than in the early days of the century. If this is so, then the notification rate should be drawn as being higher in the earlier years, that is to say, even steeper than shown here, and the error is in the right direction to support the conclusions drawn below. (It is considered by the Central Board of Health that the notification system has achieved approximately 85% accuracy fairly consistently since about 1915.)

Secondly, it may be argued that nowadays patients are being kept alive longer than was the case two or three decades ago. If this is true, that a certain number of deaths are being postponed by modern medical methods, then the real number of deaths per cases notified would be actually higher in the later years than as shown; that is, the death rate curve should not slope so steeply, and again the error is in the right direction.

Conclusions.

If it is allowed that the curves in Figure II give a fair picture of the facts, the two main points to be deduced from the percentage curve are these.

1. Of all the people in South Australia to be notified as suffering from pulmonary tuberculosis, 60% to 70% eventually die of that disease. In other words, once a patient has been notified as having the disease his chances of recovery are only one in three. These odds, of course, are modified in individual cases by such factors as the extent to which the disease has progressed when notification occurs. The fact remains that, overall, the chances of recovery are not good.

2. The percentage of deaths to notified cases has remained unchanged since 1912. The value in 1912 was 70%, whence it dropped to 61% in 1915 and thereafter it has shown if anything a slight tendency to increase, to a figure of 71% in 1944; although this latter value is most probably the crest of another shallow wave such as occurred in 1920, 1925 and 1934.

These two points in conjunction may be restated thus. In regard to pulmonary tuberculosis in South Australia, the percentage of deaths per 100,000 population in any one year to cases notified in the same year has, since 1912, remained virtually unchanged at 60% to 70%. This would seem to be of no little significance in a survey of the disease in this State, particularly in view of attempts to eradicate it. A more detailed survey, which took into consideration sex and age grouping, would shed greater light on the details of the problem; but it would appear that in the broad view all the science and money involved in the battle to save sufferers from pulmonary tuberculosis over the last thirty-five years are achieving little or no success. In other words, these figures support the contention put forward by many medical men in recent years, that, although the death rate has fallen and is falling, the outlook for the individual patient is no better now than it was thirty years ago.

The falling death rate would appear to be due not to any clinical measures, but entirely to the fall in the morbidity rate. A strong argument is deducible in favour of concentrating on preventive measures such as have been so successful in reducing the case rate to date.

It is worth repeating that the above work is something of an experimental effort and is by no means exhaustive. It may, however, serve some small purpose on its own account and perhaps a larger purpose in pointing a way to further statistical analyses of this and other diseases in this State and elsewhere.

Acknowledgements.

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(2) M. J. Holmes: "Tuberculosis in Australia", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1937, page 813.
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Reviews.

A TEXT-BOOK OF SURGERY.

ROMANIS AND MITCHINER'S well-known surgical text-book, which reflects the teaching of the famous medical school at Saint Thomas's Hospital, London, has just appeared in its eighth edition.[2] The book as a whole is a fine example of a standard English text-book, and is apparently as popular as ever in Great Britain. It is produced in two volumes, has been brought up to date in many of its sections and is well illustrated with large numbers of typical examples of disease processes.

The first part of the book deals mainly with the general aspects of surgery, and is in the main excellent. Specific diseases are dealt with very well, although leprosy is still attributed to the eating of badly cured fish. The role played by *Bilharzia mansoni* as a disease vector in Africa, in many ways just as important as *Bilharzia hæmatobia*, is completely overlooked. It would be advantageous had some fuller statements as to the modern conceptions of surgical shock been included in this section. In the discussion on septicæmia, the distinction between it and bacteriæmia is not emphasized sufficiently, while throughout the great value of the systemic administration of penicillin in local infections, such as carbuncle and cellulitis, is not stressed sufficiently. The section on surgical pathology of tumours has been rearranged with some new illustrations, and there is a good chapter, particularly on the clinical side, dealing with injuries to peripheral nerves. A little more space might have been given to hand infections, particularly on the treatment side, in which the great importance of the position of function should be mentioned. As is to be expected, the chapter

[1] For further information see the annual reports and Public Health Notes issued by the Central Board of Health of South Australia, the South Australian Statistical Registers and the Commonwealth Year Books.

[2] "The Science and Practice of Surgery", by W. H. C. Romanis, M.A., M.B., M.Ch. (Cantab.), F.R.C.S. (England), F.R.S. (Edinburgh), and Philip H. Mitchiner, G.B., C.B.E., T.D., M.D., M.S. (London), F.R.C.S. (England), D.Ch. (Durham); Eighth Edition; 1948. London: J. and A. Churchill, Limited. In two volumes, 9¼" x 6¼". Volume I: General Surgery, pp. 898, with many illustrations; Volume II: Regional Surgery, pp. 962, with many illustrations. Price: 25s. each volume.

dealing with fractures and dislocations is a fine survey of these subjects and contains all that is required. The same may be said of the sections dealing with disease of bone and deformities. At the end of this section is a good discussion of the surgical principles associated with amputation. A new feature is the introduction of special chapters on plastic surgery and on the use of X rays in diagnosis, each in its way a very fine addition to the book.

The second volume deals with regional aspects of diseases and altogether is hard to fault. This is particularly true of the sections dealing with special senses, with the breast, and with diseases of the upper part of the abdomen. The section on the thyreoid gland has been rearranged and is much improved, although the importance of modern methods in the use of thiouracil is not emphasized sufficiently. The section on intestinal obstruction is better than it was in the last edition, and a reasonable emphasis is now laid on fluid therapy and the maintenance of fluid balance. The section dealing with urological disease has been somewhat extended; some new illustrations have been added, and it is succinct and clear as regards the clinical aspects.

The book could be improved somewhat by more detailed accounts of the treatment of such conditions as burns and by the omission of the chapter dealing with arterial ligation, which, as was stated in a previous review, should be replaced if possible by a short account of the methods now used for full arterial exposure. It would be a great improvement to the whole work and would make it more interesting to students if more allusions were made to those great surgeons who have really added something to the science and art of surgery. It has also become quite a feature of some standard text-book to add at the end of every chapter a short bibliography of classical monographs or contributions, and as this is very valuable to students for reference purposes it is suggested that this would enhance this book's value.

Both volumes, as a whole, are well produced, and as they reflect the quiet progress of a great English hospital, can be recommended both to students and to practitioners as a very sound text-book of surgical practice.

ENDOCRINE THERAPY.

ELMER L. SEVRINGHAUS has provided a sixth edition of "Endocrine Therapy in General Practice" within ten years of its original publication.[1] The previous editions have served their purpose admirably and the new one is sure of a welcome if it becomes available in this country, as it will serve as a most readable record of modern advances in endocrinology.

It may be useful to refer more particularly to some of the new material introduced into the present edition. We may be sure that the author was definitely convinced of the soundness of these interpolated statements, as he has done his best to make them brief and to disturb the lines of print as little as possible. Indeed it is surprising to find how very little of the previous revision has had to be deleted or altered.

Though it is not possible to obtain pure crystalline preparations of the growth-promoting (chondrotrophic) and the adrenotrophic hormones, it remains true that most of the anterior pituitary lobe functions, though they occupy the central position, still have to be classified by the effects produced in other individual glands. Not only this endocrine complexity, but nutritional effects, genetic pattern and accidents of infection combine to produce the growth process. Indeed, we also have to recognize mixed reflexes in which some impulses are carried by nerves and others by circulating hormones.

Though certain adrenal functions appear to be carried on at a lower rate even in the absence of the pituitary, experimental evidence has been adduced to show that partial atrophy of the adrenal cortex follows removal of the pituitary, and that adaptations to stress can no longer be achieved by the animal. Adrenal cortical functions include the synthesis of certain proteins, such as the globulins, which are related to immune reactions to infection. The pituitary responds directly to adrenal medullary action, but inversely to adrenal cortical activity. Stimulation of the adrenal cortex by pituitary extracts or by stress situations causes remarkable diminution in the concentrations of cholesterol and ascorbic acid in the adrenal tissue. It is thought that cholesterol is converted into cortical hormone, but the utilization of ascorbic acid is still unexplained.

Adrenal function concerned with preservation of optimal blood volume and its potassium and sodium concentrations may be tested in several ways. If the result of the water diuresis test is equivocal, the standardized test of concentration of urinary chloride, which is more rigorous, though more hazardous, is recommended. Negative results are dependable in excluding the diagnosis of Addison's disease.

Increase in excretion of uric acid on administration of adrenocorticotrophic hormone from the pituitary is the basis of another tolerance test involving the adrenal functions concerned with carbohydrate and protein metabolism which are not involved in the tests for Addison's disease. Experimental evidence has also been adduced to justify the belief that the adrenal medulla is concerned with prompt mobilization of resources for meeting many stress situations, and that the adrenal cortex can stimulate the provision of greater resources for future stresses.

The relative potencies of adrenal cortical extracts cannot be determined exactly because of the variable proportions of two or more active hormones. The difficulty of freeing cortical preparations from epinephrine-containing elements of the medulla further complicates the problem of the manufacture of reliable active assayed cortical products for oral administration.

The implantation of synthetic hormone materials in pellet form has reached the stage of practicability; but the patient's daily requirement should first be estimated by means of injections of the drug in oil, and if toxic symptoms become apparent, surgical removal of the implanted pellet must be undertaken.

In the chapter on the thyreoid a slight rearrangement has been made, the account of thiouracil therapy being brought into its proper sequence. In another place an unconvincing statement is made that there may be indications that the thyreoid can inactivate an excess of thyreoid hormone. In non-toxic goitre adults, thyreoid should be tried, though the use of iodine for this purpose is seldom successful after the age of twenty years.

In the discussion of the treatment of tetany, a note has been added as a warning not to attempt long-sustained use of the special low-phosphorus diet mentioned.

Only two short paragraphs have been added to the long chapter on obesity. Reference is made in one of them to the Yale experiments on the effects of destructive lesions in the hypothalamic area. In the other paragraph "Benzedrine" is defined as useful in the treatment of obesity when the dietary regimen is first restricted. It has the effect of counteracting the usual increased hunger, but this benefit is not maintained for more than a few weeks.

The author has introduced into this edition quite a large number of important advances in knowledge concerning the interrelationship of the anterior lobe of the pituitary gland and the gonads. Œstradiol, manufactured in the ovarian follicle, is known to be much more potent than œstrone, which has been found in the ovary. The liver is active in several steps in the metabolism of œstradiol, including the excretion of œstrogenic material in the bile, leading to reabsorption and further metabolic changes. Not more than 20% of progesterone, the *corpus luteum* hormone, is excreted as pregnandiol, conjugated with glycuronic acid, the assumption being that most of the progesterone is metabolized to products as yet unknown. Normal oxidative products of œstradiol and œstrone stimulate the pituitary to produce the hormones which lead to progesterone secretion; this deviates the metabolism of œstradiol and œstrone towards œstriol, which cannot stimulate the pituitary in this direction. In this manner *corpus luteum* formation occurs, its activity is terminated, menstruation takes place and the cycle of actions is repeated; but menstruation itself is still awaiting definition. It has been found that large doses of chorionic gonadotrophin (prolan) will sustain the secretion of progesterone by a *corpus luteum* in a non-pregnant woman for several weeks.

It is well known that great care should be taken to avoid large dosage with œstrogens and progesterone because of the disturbing effects on the menstrual cycle, but in pregnancy it has been demonstrated that large dosage is safe when used to combat toxæmia or to develop the breasts. The reason is that normally in pregnancy there is a great increase in the amounts of both types of hormones. When œstrogens are being employed in climacteric therapy, the addition of progesterone (ten milligrammes or more daily for not more than a week at a time) will alleviate the disagreeable tenderness and swelling of breast tissues without interfering with the beneficial effects of the œstrogens. There is no fear of initiating carcinoma with the ovarian hormones; but they provide catalytic stimulation for more rapid growth after the carcinogenic factor has started the process.

[1] "Endocrine Therapy in General Practice", by Elmer L. Sevringhaus, M.D., F.A.C.P.; Sixth Edition; 1948. Chicago: The Year Books Publishers, Incorporated. 8" x 5½", pp. 264, with many illustrations. Price: $4.00.

The abrupt fall and rise in daily basal temperatures which are believed to occur about the time of ovulation and the beginning of the function of the *corpus luteum* may be used as a guide to help in the detection of failure of ovulation or luteal function. It may be possible even to indicate the optimal day for fertilization or for hormonic treatment by this simple technique.

An osteoporosis of generalized type, which may be associated with such deformities as compression of the vertebræ, may be indicative of great depression of ovarian function, which is amenable to intensive and prolonged œstrogen therapy.

Œstrogenic therapy is no longer used for gonorrhœal vaginitis, as sulphonamide or penicillin treatment has become more economical and more satisfactory.

The attention of the medical profession is drawn to the efficacy of percutaneous œstrogenic therapy, which has been demonstrated by the value of cosmetics containing œstrogens in stemming the progression of certain senile changes. An improvement has also been made in tablets for oral use, with the preservation of much greater biological activity than was hitherto possible. Comparable progression has also been made with synthetic substances to be used orally for climacteric treatment.

Progesterone, in addition to being useful for the relief of dysmenorrhœa and for the prevention of repeated abortion, may now be used in the treatment of premenstrual tension and painful engorgement in the breasts. In the prevention of repeated abortion the dosage has been substantially increased, and it is recommended that 20 to 50 milligrammes should be given daily with gradual reduction and withdrawal at the end of the fourth month of gestation; when that stage is reached we may depend on the placental production of progesterone.

Testosterone is usually prescribed as the propionate; but an aqueous suspension of microcrystals of free testosterone has been used with comparable effect, and pellets for implantation should soon be available. It is likely that the chief testicular inhibitory effect on the anterior pituitary secretion of gonadotrophic hormone is exerted by a hormone from normal spermatogenic tissue which has not yet been obtained in a pure state.

It is now believed that there is no truly pancreotrophic hormone, but that other hormones produced by the pituitary can cause exhaustion of island tissue with the production of *diabetes mellitus*. Administration of certain steroids believed to have adrenal cortical activity will produce diabetes in laboratory animals. Insulin facilitates the combination of glucose with phosphoric acid by neutralizing the effect of substances found in anterior pituitary extracts which would otherwise be free to interfere with the functioning of hexokinase as a catalytic agent.

A six-page section has been added to the chapter on *diabetes mellitus*, in which the author stresses the desirability of prescribing vitamin supplements to the dietary. He favours the use of multivitamin concentrates, as they provide the greatest assurance of adequacy.

The excellent illustrations are exactly the same as those in the previous edition. We can strongly recommend this book for careful study by all practising physicians.

FINAL EXAMINATIONS IN MEDICINE.

Of recent years textbooks in which the subject matter is arranged alphabetically rather than systemically are becoming increasingly popular with medical students, and such a book is "Essentials for Final Examinations in Medicine", by John de Swiet.[1] Whilst this little book is not the alpha and omega of medical handbooks, yet all students will find that its pocket-size compactness makes it a most useful companion to any of the larger textbooks of medicine. The ætiology, clinical features, diagnosis, prognosis and treatment of all medical maladies from achalasia to uræmia are clearly but briefly discussed, and the dogmatic manner in which each topic is dealt with enables the student to obtain a clear picture which he should be able to reproduce at examination time.

The fact that the third edition now appears only six years after the first adds its own recommendation to this work which can be read with advantage by all medical students. Tutors, too, will find many aids to the teaching of medicine herein.

[1] "Essentials for Final Examinations in Medicine", by John de Swiet, M.D. (London), M.R.C.P.; Third Edition; 1947. London: J. and A. Churchill, Limited. 7¼" × 5", pp. 184. Price: 9s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The British Pharmacopœia, 1948", published under the direction of the General Council of Medical Education and Registration of the United Kingdom. London: Constable and Company, Limited. 9" × 6", pp. 954.

"The Victorian Coroners' Manual", revised by Irvine W. Williams; 1947. Melbourne: Issued by the authority of the Hon. W. Slater, His Majesty's Attorney-General and Solicitor-General. 11" × 8½", pp. 88. Price: 10s.

"Inside Red Russia", by J. J. Maloney, M.L.C.; 1948. Sydney and London: Angus and Robertson. 8¼" × 5½", pp. 214. Price: 4s. 6d.

"Midwifery", by Ten Teachers, under the direction of Clifford White, M.D., B.S. (London), F.R.C.P. (London), F.R.C.S. (England), F.R.C.O.G., edited by Clifford White, Frank Cook and William Gilliatt; Eighth Edition; 1948. London: Edward Arnold and Company. 8½" × 5½", pp. 568, with many illustrations. Price: 20s.

"Tuberculosis: A Discussion of Phthisiogenesis, Immunology, Pathologic Physiology, Diagnosis and Treatment", by Francis Marion Pottenger, A.M., M.D., LL.D., F.A.C.P.; 1948. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9½" × 6½", pp. 598, with illustrations. Price: 90s.

"The Mechanism of Abdominal Pain", by J. V. Kinsella, M.B., Ch.M. (Sydney), F.R.C.S. (England), F.R.A.C.S.; 1948. Sydney and London: Angus and Robertson, Limited. 9½" × 6", pp. 246, with illustrations. Price: 32s. 6d.

"The 1947 Year Book of Neurology, Psychiatry and Neurosurgery"; Neurology—edited by Hans H. Reese, M.D., and Mabel G. Masten, M.D.; Psychiatry—edited by Nolan D. C. Lewis, M.D.; Neurosurgery—edited by Percival Bailey, M.D.; 1948. Chicago: The Year Book Publishers Incorporated. 7" × 4½", pp. 702, with many illustrations. Price: $3.75.

"Medical Research in War", Report of the Medical Research Council for the Years 1939-45; 1948. London: His Majesty's Stationery Office. 9½" × 7", pp. 456. Price: 7s. 6d. net.

"Tuberculosis in Young Adults: Report on the Prophit Tuberculosis Survey, 1935-1944" (Royal College of Physicians), by M. Daniels, M.D., D.P.H., F. Ridehalgh, M.A., M.B., B.Ch., M.R.C.P., and V. H. Springett, M.B., B.S.; 1948. London: H. K. Lewis and Company, Limited. 10" × 7¼", pp. 244, with 80 illustrations, some of them in colour. Price: 30s. net.

"The Anatomy of the Eye and Orbit, Including the Central Connections, Development and Comparative Anatomy of the Visual Apparatus", by E. Wolff, M.B., B.S. (London), F.R.C.S. (England); Third Edition; 1948. London: H. K. Lewis and Company, Limited. 10" × 7½", pp. 448, with 323 illustrations, many of them in colour. Price: 45s. net.

"Conference on Metabolic Aspects of Convalescence: Transactions of the Fifteenth Meeting", edited by Edward C. Reifenstein, junior, M.D.; 1947. New York: Josiah Macy Jr. Foundation. 9" × 5¼", pp. 164, with illustrations. Price: $2.25.

"Public Health Administration in the United States", by Wilson G. Smillie, A.B., M.D., Dr.P.H., Sc.D. (Hon.); Third Edition; 1947. New York: The Macmillan Company. 9¼" × 6", pp. 658. Price: 32s. 6d.

"A Pocket Gynæcology", by S. G. Clayton, M.D., M.S. (London), F.R.C.S. (England), M.R.C.O.G.; 1948. London: J. and A. Churchill, Limited. 7¼" × 5", pp. 118, with illustrations. Price: 7s. 6d.

"Textbook for Midwives", by Wilfred Shaw, M.A., M.D. (Cantab.), F.R.C.S. (England), F.R.C.O.G.; 1948. London: J. and A. Churchill, Limited. 7¼" × 5", pp. 698, with illustrations. Price: 12s. 6d.

"Handbook of Practical Bacteriology: A Guide to Bacteriological Laboratory Work", by T. J. Mackie, C.B.E., M.D., LL.D., D.P.H., and J. E. McCartney, M.D., D.Sc.; Eighth Edition; 1948. Edinburgh: E. and S. Livingstone, Limited. 8½" × 6¼", pp. 632, with some illustrations. Price: 25s. net.

"The Problems of Family Life: An Environmental Study", by Agatha H. Bowley, Ph.D.; Second edition; 1948. Edinburgh: E. and S. Livingstone, Limited. 7½" × 5½", pp. 144, with illustrations. Price: 6s. net.

"Bilharzial Cancer: Radiological Diagnosis and Treatment", by Mahmoud Ahmed Afifi, M.B., Ch.B. (Cairo), M.R.C.S. (England), L.R.C.P. (London), D.M.R.E. (Cambridge); 1948. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 118, with 60 illustrations. Price: 16s. net.

"Sexual Endocrinology of Non-Mammalian Vertebrates", by L. H. Bretschneider and J. J. Duyvené de Wit, in collaboration with S. Dudok de Wit, B. Van Egmond, M. A. Goedewaagen, H. Heintzberger, J. Van Iersel, Chr. Jaski, E. Van Koersveld, I. Kristensen, J. Meltzer, G. J. Van Oordt and C. Pelsma, from the Laboratory for General Zoology, University of Utrecht; 1947. New York, London, Amsterdam, Brussels: Elsevier Publishing Company, Inc. 8" × 6¼", pp. 156, with many illustrations. Price: 15s. net.

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE PREVENTION OF BLINDNESS.

> Or what king, going to make war against another king, sitteth not down first, and consulteth whether he be able with ten thousand to meet him that cometh against him with twenty thousand? [ST. LUKE xiv: 31.]

No one will deny that blindness is one of the greatest enemies of mankind, if not the greatest. Anyone who questioned this statement would do well to read the presidential address delivered by Dr. J. Ringland Anderson to the Ophthalmological Society of Australia (British Medical Association) and published in *The Transactions* of that body for 1946. If this discussion is restricted to physical blindness there will be sufficient reason to claim that the subject is worthy of the closest attention. It is important to remember that blindness may be spiritual as well as physical and that some of the really blind in this world have retinæ, optic nerves and cerebral cortex which allow their possessor to see the people and things around them. Admittedly medical men and women have as their goal the ultimate welfare of man as a whole; though they would that they were able to give man full perception, or spiritual insight, as well as to preserve his physical sight, they must direct their energies primarily to the latter object—they alone in the community are trained to this end. When most people think of blindness and the blind they are influenced by humanitarian motives of sympathy for a fellow man in distress. But as has been pointed out on a previous occasion in these columns, this common and commendable attitude of mind does not exclude other points of view. The social economist is concerned with the State and with community welfare and wishes to see everyone around him able to take his place in community life as a self-supporting unit. Those whose training and interests include preventive medicine—and they will comprise all practitioners of medicine—study the causes of blindness that they may prevent its occurrence.

When we talk of the prevention of blindness it is necessary to know what the causes of blindness are and to take measures to combat them. This is a haphazard method of attack. What is needed is a definition of blindness and a survey of the whole community to discover the incidence of the various factors that may result in blindness. This means that the strength of the enemy is discovered before war with him is undertaken. Clearly factors that cause blindness may become arrested and impairment of vision result instead of blindness. If the arrest of the factors is the result of deliberate and planned effort, the outcome is the more to be prized on that account. Blindness may be the result of many conditions. These may be found set out in cold print in any good text-book of ophthalmology. Those who prefer to know how blindness from varying causes is met in everyday practice in Australia, will find a valuable contribution on the subject from the pen of J. Ringland Anderson in THE MEDICAL JOURNAL OF AUSTRALIA for November 4, 1939, at page 680. Anderson described four groups of causes: (*a*) congenital and developmental, (*b*) infectious and bacterial, (*c*) traumatic and chemical, and (*d*) causes associated with general diseases; he had a small group in regard to which no information was available. Each of the four groups had as members from seven to twelve conditions. Not all of the causes may be easily prevented. We have only to think of such conditions as *retinitis pigmentosa* and hereditary optic atrophy on the one hand and of *ophthalmia neonatorum* and industrial trauma on the other to realize how the difficulties of prevention may vary. In regard to certain causes, particularly those associated with general diseases, it is safe to assert that no preventive measures would be effective. It may also be held that the incidence of *ophthalmia neonatorum* and trachoma is in some degree an index of the extent to which the principles of preventive medicine are understood in the community and practised. To define blindness is not easy, particularly if we are concerned with blindness in industry. One discussion that is readily recalled took place at the one hundred and third annual meeting of the British Medical Association at Melbourne in 1935. Here the late James W. Barrett, discussing the definition and causes of blindness, referred to the decision of the Hobart Congress of 1934 that blindness meant inability to count fingers at a distance of one metre in any circumstances. (This decision was made when L. J. C. Mitchell brought before the Section of Ophthalmology the definitions accepted by the Victorian Branch of the Association in 1933.) Barrett was concerned chiefly with the gap between blindness and vision so defective as to render the subject unable to follow any occupation requiring vision. This gap covers a range of partial blindness (or as Barrett preferred to call it, partial sightedness) that needs careful definition. Partial blindness was agreed at the Hobart Congress to be the possession of vision of $^{6}/_{60}$ or less in any circumstances; it was also thought that nystagmus or contraction of the field might involve in odd cases the possession of a somewhat higher standard. One reason for the need of definition will be seen in the industrial law courts after industrial accidents and another possibly among children who are to be selected for attendance at a sight-saving school.

Some ophthalmological survey work has already been undertaken in Australia, but, presumably on account of the intervention of the war, little more than a start was made. In February, 1937, J. B. Hamilton and W. D. Counsell submitted to the National Health and Medical Research Council the results of a survey on the cause

and prevention of blindness in Tasmania. This was followed by a supplementary report from the same two authors which was published in this journal on March 18, 1939. Hamilton and Counsell concluded that at least 40% of the blindness in Tasmania was preventible. Some ophthalmologists have held that the Tasmanian authors' estimate was on the conservative side, and it is true that in other places higher estimates of preventibility have been made, but that does not matter a great deal. What is important is that certain factors have been found to operate which might be prevented from operating. After the presentation of the report from Hamilton and Counsell the National Health and Medical Research Council was asked to extend its grant so that a survey of the whole of Australia might be carried out. This was not done, presumably, as already mentioned, because the war intervened. The time has now arrived when the Australia-wide survey should be instituted, and it is to be hoped that an approach will be made in the near future to the National Health and Medical Research Council to enable this to be done. To take steps that will conserve the eyesight of the Australian community must surely be a task which the Council, with national funds at its command, cannot shirk. If necessary the Government should make the Council a special grant to extend over a stated period of years for this purpose. In 1950 the International Congress of Ophthalmology will be held in London, and one of the actions of this assembly will be to discuss the prevention of blindness and to lay down a nomenclature for use in all parts of the world. If an Australian survey is in the making when this international gathering is held, Australian ophthalmologists should be able to offer a worthwhile contribution. But this London meeting in 1950 is really an additional reason for the inauguration of a survey without delay. What has to be kept continually in mind is that blindness is a relentless foe to be opposed with every available weapon. The foe will be the more readily overthrown if his forces are known and plans for his defeat are made with calm deliberation before he has time to claim further victims.

Current Comment.

ERYTHEMA MULTIFORME EXSUDATIVUM.

THE condition known as *erythema multiforme* is not quite so rare as is sometimes stated. Ex-service medical officers report that examples of the condition were continually appearing in different units during the last war, and that the number of cases, if collected, would not have been small. It is an interesting condition because of its obscure origin, if for no other reason. Varieties of *erythema multiforme* are named according to the more prominent features of the lesions composing the eruption. The form known as *erythema multiforme exsudativum* is familiar in medical practice. Molesworth states that it occurs in spring or autumn and shows a considerable tendency to recurrence, generally once or twice a year. Sometimes a severe form of the condition is accompanied by extensive involvement of the mucous membrane. In a report published in 1946[1] the Commission on Acute Respiratory Diseases in America described six cases of *erythema multiforme exsudativum* with predominant involvement of the mucous membrane, and it paid particular attention to the association of pneumonia with the condition.

Erythema multiforme exsudativum was described as a specific entity by Hebra in 1860. Harry Keil states[1] that the importance of Hebra's endeavours lay primarily in the assembling of apparently diverse cutaneous lesions under one head and in the recognition of the principle that they represented phases of a single condition. Keil explains that the qualifying word "multiform" was probably first used by Hebra for the purpose of indicating the close relationship existing between several dermatological lesions which had previously been classified in separate categories. He goes on to state in regard to polymorphous eruptions that other diseases such as *dermatitis herpetiformis* and acute *lupus erythematosus* were also seen to be accompanied by lesions that were just as multiform in appearance as those in *erythema multiforme exsudativum*, and that despite this the term *erythema multiforme* seems destined to be retained in nomenclature, its meaning being variously restricted or broadened.

Mention has been made of the obscure origin of the condition. Norman Walker and Percival state in their textbook that the development of the skin lesions indicates some form of poisoning and that it is to the detection of its source that the efforts of the physician should be directed. They think that some article of diet may be responsible and that a search should be made for such an offender. Molesworth states that the disease seems to be of toxic origin. He thinks that in patients who are subject to regular or frequent recurrence the extirpation of the tonsils as a possible focus of infection is worthy of consideration and that success has not infrequently followed the adoption of such measures. He adds that in other cases the tonsils are apparently not affected. The lesions, in his opinion, are so true to a definite type that the disease is more probably due to a specific infection than to absorption from a focus of sepsis due to common organisms. Under such an hypothesis the tonsils or other lymphoid tissue or the crypts of such organs may be merely the hibernating places in which a nidus of infection persists between outbreaks. This would explain the occasional success that follows extirpation of the tonsils. Vague and hypothetical as this explanation is, Molesworth thinks that it is the best available at the moment.

The Commission on Acute Respiratory Diseases obtained inconclusive results from bacteriological studies. It states that some authors have been impressed by the possible causal significance of streptococci recovered rather frequently from cultures from the mouth and throat. In one fatal case streptococci may have played a role, since they were recovered in pure culture from bullæ. The Commission points out, however, that other observers have not succeeded in growing organisms from bullæ. The Commission thinks that it is apparent that hæmolytic streptococci are not found with enough consistency in the majority of cases to suggest their causal relationship to the disease; in occasional instances they may play a contributory or secondary role. Keil, in his discussion on ætiology, refers to a possible virus causation. He also mentions possible factors of food and sunlight. He is averse to the acceptance of a multiple ætiology. He thinks that the data available to him indicate that the disease is a well characterized identity in the sense of rheumatic fever as contrasted with "rheumatism". He quotes words used by Hebra in 1860 as follows: "These erythemata are often ascribed to catching cold, or to errors in diet, or to mental emotions; but unless the real existence of these conditions can be proved, I regard such expressions as mere commonplaces and shibboleths; and, rather than avail myself of them, I shall confess that the cause of these diseases is altogether unknown to me. It is certain that they do not owe their origin either to the imbibition of alcoholic liquors, or to eating any particular type of food, whether sour, sweet or bitter, whether of animal or vegetable nature". We must agree with Keil that after a lapse of over 75 years substantially little can be added to Hebra's statements.

[1] *Archives of Internal Medicine*, Volume LXXVIII, 1946, page 687.

[1] *Annals of Internal Medicine*, Volume XIV, 1940, page 449.

The pathology of the condition is explained by Sequeira, Ingram and Brain in their textbook. They state that the essential skin changes are due to the action of circulating toxins upon the small vessels or upon the nerves controlling them; this results in dilatation and the exudation of plasma, the latter causing œdema of the prickle cell layer and forming vesicles or blebs at various levels in the epidermis. A cellular infiltration is most marked about the dilated vessels and may reach the deepest layers of the corium and also invade the epidermis, making the vesicles and blebs cloudy and purulent. Erythrocytes may escape from the vessels and colour the lesions.

M. Finland, L. S. Joliffe and F. Parker, junior, have recently published a clinical study[1] of pneumonia and *erythema multiforme exsudativum* which is of interest from the ætiological point of view. These authors describe their cases in some detail and they reproduce photographs of the lesions with skiagrams of the chests and photomicrographs of the lesions. The lesions were those commonly regarded as *erythema multiforme exsudativum*. Three of the patients died. The pneumonia of three of the four patients (one of the three was the one who recovered) was identical with severe primary atypical pneumonia with extensive bilateral miliary type of involvement. In the fourth case, though the symptoms and signs were consistent with the presence of pneumonia, autopsy revealed mostly congestion and œdema. In three of the four cases there was evidence suggestive of a possible infection with a psittacosis-like virus. No virus was actually isolated. In a footnote it is stated that after the paper was submitted for publication there was isolated in the fourth case a virus serologically related to herpes. It is also stated that the exact relation of this virus to the pulmonary and muco-cutaneous lesions is still uncertain.

Observations such as those of Finland and his co-workers are important and show how light may be shed on this peculiar condition. The clinical side has scarcely been mentioned. It must suffice in this regard to state that such drugs as "Benadryl" and "Rutin" which have an antihistamine action have been used and that beneficial results have followed their exhibition in some cases.

THE DISPOSAL OF WASTE FROM RAILWAY CARRIAGES.

IT seems that in the United States, as in Australia, the basic principle in the disposal of human wastes from railway passenger carriages can be summed up in the old adage: "Out of sight, out of mind." From time to time since as far back as 1899, according to Abel Wolman and Lloyd K. Clark,[2] enthusiastic individuals have raised the æsthetic and public health issues involved, but nothing very constructive has been done. It has been assumed by most people concerned (though never verified by any epidemiological investigation) that current practice constitutes a menace to health, but a practical alternative to the crude disposal of waste material onto the railway tracks has been hard to devise. After much inconclusive discussion a research project was finally approved by the Association of American Railroads and this got under way in 1946 with Wolman, who is professor of sanitary engineering at the Johns Hopkins University, as consultant director and Clark as project manager.

The first question considered was the assumption that the disposal of sewage along railways was a significant factor in disease, and for this they called on an epidemiologist, Kenneth F. Maxcy, who reviewed the relevant evidence from 1900 to 1945. Maxcy's report concludes:

It can be stated with reasonable assurance that information at present available fails to establish the existence of a public health menace resulting from the method of disposal of fecal wastes employed by railways. This by no means proves the negative—that such a menace does not exist. It is reasonable to assume, however, that this practice has in the past been a relatively unimportant route of dissemination of the pathogenic organisms which cause the commonly recognized enteric infections.

Another essential aspect which had never been investigated related to the habits of passengers in the use of toilets and the nature and quantity of the waste materials. This was undertaken with the thoroughness characteristic of much American research and a remarkable collection of data obtained. The toilet habits of more than 2000 persons were studied while they were travelling a total of 6251 passenger-hours over a total distance of 320,000 passenger-miles. The average number of entries per hour, the number of passenger-hours per entry and the average time of occupancy have all been relentlessly recorded with a number of other details that do not need consideration here. One cannot but remark, however, that the passengers must have been either blissfully ignorant of what was going on or remarkably lacking in self-consciousness. Of more obvious relevance to the main problem are the data obtained concerning the nature and quantity of waste material. The sewage was analysed according to standard procedures and in addition the nature and number of foreign objects (which ranged from a miniature whisky bottle to orange peel) were determined. A more extensive survey of foreign objects was made by means of a *questionnaire* sent to 29 railroad companies; this revealed a list of 58 different types of objects which had caused blockages, ranging from radiator caps to human fœtuses. The total sewage solids deposited from the test cars to the railway track per average hour were estimated at 0·121 pound dry weight. The average yearly deposition per mile of track from a single coach is calculated at 0·858 pound dry weight, though the heavily loaded trains between New York and Washington gave amounts three times as great. It is further calculated that all trains in the United States release annually a total of 3,460,000 pounds dry weight of sewage solid, which works out at 0·302 ounce dry weight per yard of track per year. The actual amounts thus appear insignificant, but the distribution is by no means uniform, the quantity on the New York to Washington run being worked out at 1·067 pounds dry weight per yard per year and an actual examination of part of the track revealing that on the average there were evidences of 13 discharges per mile containing recognizable fæcal matter. Bacteriological examination of ballast which had been used on a busy track for about ten years revealed the presence in 50% of surface ballast of approximately four coliform organisms per square centimetre of surface of stone, no organisms being grown from the other 50%, and in subsurface ballast of coliform organisms in all samples, 80% of samples having counts of fewer than 50 per square centimetre of surface area. No organisms were grown from samples of new unused ballast.

These investigations still leave the public health significance of the problem uncertain, though it can surely not be ignored; there is certainly an æsthetic aspect, at least on the busier lines. Wolman and Clark report that a great many treatment methods and devices have been investigated, the main results sought being comminution and disinfection. A device for comminution must be rugged to deal with metal and glass objects; it must be small and a low consumer of power. The search is still continuing, but one such unit is already in use with as yet satisfactory results. Disinfection of macerated toilet wastes by heat has been found to be quite practicable, either steam or electricity (or a combination) being a satisfactory source; a temperature not exceeding 160° F. and a holding time of not more than a few seconds will destroy the coliform organisms. Simpler devices are being designed for branch lines, and it is suggested, probably with justification, that the old-fashioned system is quite satisfactory for long runs through unpopulated areas, but active steps are being taken to develop treatment units for busy lines in populated areas. This activity is certainly not before its time, whatever may be one's views on the epidemiological aspect. It would be interesting to know whether this matter has received attention in our own country.

[1] *The American Journal of Medicine*, April, 1948.
[2] *American Journal of Public Health and The Nation's Health*, May, 1948.

Abstracts from Medical Literature.

RADIOLOGY.

Cholesteatomata of the Temporal Bone.

H. K. GRAHAM HODGSON (*British Journal of Radiology*, May, 1947) states that a cholesteatoma of the mastoid requires two conditions for its formation: firstly, a low-grade, chronic infection, and secondly, a diploetic or poorly pneumatized mastoid. The condition is associated with a long-standing history of a copious and foul aural discharge. In a cellular mastoid, the infection spreads from the middle ear, through the *aditus ad antrum* into the mastoid antrum, and thence into the mastoid cells. In an acellular mastoid, if the infection is of some virulence, the diploetic bone is infiltrated by the organisms and diffuse bone infection takes place; but if the invasion of the acellular mastoid is by an organism of low virulence, then a much slower process takes place. The normal mucosa of the middle ear is destroyed and gradually replaced by epidermal cells, either by extension of the epidermis from the external auditory canal through the ruptured drum or by cellular metaplasia. These epidermal cells are continually being desquamated by the infection and replaced, and they collect in a slow but constantly enlarging mass in the epitympanic recess, the *aditus ad antrum* or the mastoid antrum. The organism is not sufficiently virulent to infiltrate to any extent the surrounding acellular bone, but it gradually softens it by infection and erodes it by the pressure of the constantly enlarging mass of dead cells. A thin, protective ring of sclerosed bone is frequently seen around the expanding "tumour", but when the mastoid is of the sclerotic type, or when a good deal of sclerosis has taken place surrounding the cholesteatoma, this ring may be concealed in part or entirely. It is obvious that until the pseudo-tumour has eroded the walls of the cavity it occupies appreciably beyond the normal limits, there will be no radiological evidence of its presence. Frequently, because of its comparatively small size, the signs of erosion will first be seen in the *aditus ad antrum*. As the cholesteatoma erodes more and more bone, a time may come when perforation of the containing wall occurs. One sees an area of lesser density than the surrounding bone, but not so transradiant as a solitary air cell of the same size would be. It is usually in the immediate neighbourhood of the mastoid antrum or attic, and it may encroach on or perforate the posterior wall of the external auditory canal. This area of bone destruction is frequently smooth in outline and of oval or rounded shape, but it may occasionally be irregular in shape, particularly in those mastoids in which a few scattered cells are present. In the postero-anterior oblique projection this area should be looked for in the neighbourhood of the apex of the external canal. In the lateral oblique view it is seen in the area bounded above by the *tegmen tympani*, and in front by the auditory canal. Perforation of either of these structures may be shown as actual gaps in the bone, but more usually as areas of local rarefaction. A cholesteatoma is distinguished radiologically from a mastoid abscess, firstly, by its location in the immediate neighbourhood of the antrum or attic, whereas an abscess is more common in the peripheral zone; secondly, a cholesteatoma occurs almost invariably in an acellular or poorly pneumatized mastoid, whereas an abscess may occur in either a cellular or diploetic mastoid, but more commonly in the former. Cholesteatoma is distinguished from a solitary air cell by its location, by the fact that examination of the opposite mastoid will reveal no corresponding cell, and also by the fact that the bone surrounding a cholesteatoma shows reactive changes, and a cholesteatoma is usually more opaque than an uninfected cell of corresponding size would be.

Ankylosing Spondylitis.

G. L. ROLLESTON (*The British Journal of Radiology*, July, 1947) states that the earliest recognizable appearance of ankylosing spondylitis occurs first in the sacro-iliac joints, and then, after a short interval, characteristic changes are seen at the periphery of the vertebral bodies and in the costo-transverse articulations. The earliest sacro-iliac changes may be described as a patchy persistence of the adolescent features of the normal joint, seen in adult life when such appearances would not normally be expected. The normally well-defined subarticular cortex of the articular surfaces is absent. The joint margins show a vignetted appearance. These changes are attributable to small juxtaarticular areas of erosion which absorb the subarticular cortex and the adjacent underlying cancellous bone. Close scrutiny should be given to the superior vertebral angle of the ilium where a ragged, ill-defined edge, due to loss of the superficial cortical layer, is very significant. As these areas of erosion become larger, the juxta-articular bone shows a typical "moth-eaten" appearance. There is widening of the joint space, which is more apparent than real and is accounted for by the subarticular bone erosion. These appearances are more readily seen in the upper third of the joint. The next stage of sacro-iliac change is progressive loss of joint space due to destruction of the hyaline and fibro-cartilage. Associated with the loss of joint space there appear the characteristic mottled amorphous calcium deposits in the juxtaarticular portion of the ilium, and especially in its lower third. In the final and quiescent stage of the disease these amorphous calcium deposits are completely absorbed and the bony cancellous architecture is reconstructed. Final and complete bony ankylosis occurs at the sacro-iliac joints. The pathological changes in the spinal column in ankylosing spondylitis are usually described as progressing in a caudo-cephalic direction. In the author's series of cases, this mode of spread has been found to be more the exception than the rule. The earliest vertebral body changes have been recognized in the upper lumbar region. Isolated changes have also been noted in the cervical bodies. This would indicate that portions of the spine may be by-passed initially, and this fact is of considerable importance. Early changes in the vertebral bodies are most readily seen in the lumbar region and are recognized in the lateral projection; there is distinct sharpening with loss of normal convex contour of the anterior margins of the vertebral bodies, and there is a "filling in" of the normal concavity of the anterior surface of the body. The combination of these two effects produces a "squaring" of the vertebral body. These appearances must be attributable to early changes in the anterior common ligament and must represent the precalcification stage that leads on to the classical "bamboo spine". Ankylosing spondylitis involves the costo-transverse joints at a comparatively early stage and results in a great diminution of chest expansion. The articular surfaces show irregular "moth-eaten" erosions and local osteoporosis. In the advanced stages of the disease complete bony ankylosis will occur. The recognition of these changes should be immediately followed by an examination of the sacro-iliac joints. In some cases they may constitute the first indication of the true nature of the disease from which the patient is suffering. If the disease is recognized and adequately treated by X-ray therapy at an early stage, the patient may be almost completely restored to normal health.

Cranial Manifestations of Fibrous Dysplasia of Bone.

FRANK WINDHOLZ (*American Journal of Roentgenology*, July, 1947) states that the X-ray diagnosis of fibrous dysplasia of the skull is based on the morphology of bone changes. It seems that the distribution, extent and appearance of the newly formed bone tissue follow specific rules. These are influenced by the vascularization of connective tissue and by the statics of the cranium which differ from those in other parts of the skeleton. The base of the skull, which is subjected to greater stress than the calvarium, produces more bone tissue. Consequently, it is dense in radiographs. Later, however, this tissue grows not only according to its adaptation to static principles, but also as a result of intrinsic factors of osteogenesis. Bone formation, often following the course of the greater blood vessels and their well-defined borders, produces sharply outlined densities. In radiographs these alternate with translucent areas of connective or non-calcified osteoid tissues. The presence of dense bone spicules and alternating clear, translucent areas of ground glass quality are characteristic radiological findings. Visualization of them permits not only the correct diagnosis to be made, but differential diagnostic considerations of cranial manifestations of radiologically similar systemic bone diseases as well. In von Recklinghausen's disease, cystic changes in the skull are associated with characteristic, diffuse, granular decalcification of the entire cranium. Consequently, if the general background remains normal in appearance, the cystic changes cannot be considered due to von Recklinghausen's disease. In xanthomatosis, Schüller-Christian's disease, and other reticulo-endothelial disorders, no true cysts are observed. Defects of the vault do not possess their own bony wall; they are simple, large, sharply outlined, and often multiple holes. In both von Recklinghausen's and Schüller-Christian's diseases, the clinical picture is entirely different from that of fibrous dysplasia. Solitary xanthomata and other bone defects of

the skull do not produce appreciable amounts of bone; consequently, as a rule, they do not reveal internal bone structures. In Paget's disease, the margins between normal and transformed bone are mostly not sharply outlined; transition is continuous. The cotton wool appearance of the bones of the skull in Paget's disease usually reveals rounded, indistinctly outlined densities. They are surrounded by coarse, trabeculated residuals of spongy structures of the diploë. Larger translucencies of ground glass type are absent. In addition, the disease produces a relatively uniform thickening and, in advanced cases, characteristic enlargement of the cranial vault. This seems to be the result of softening of the newly formed Paget bone. It involves large areas of the cranium. Usually there is no circumscribed bulging or expansion of the bones. If the entire cranial vault is involved by Paget's disease, the radiological changes are so characteristic that they cannot be confused with the irregular, sharply outlined spicules, densities, and cyst-like translucencies of fibrous dysplasia. Bony reactions of meningiomata are progressively growing, solid thickenings of the vault. Destructive bone lesions caused in rare instances by meningioma are, in contrast to foci of fibrous dysplasia, not surrounded with continuous bone margins. Fibrous dysplasia starts in early youth. Bony reactions of meningiomata occur, as a rule, in adults. They involve first the inner table; diploë and outer table are changed later. In fibrous dysplasia the diploë is the primary site of localization of the disease.

Calcification in Suprarenal Neoplasms.

ERIC SAMUEL (*The British Journal of Radiology*, March, 1948) states that of the malignant suprarenal tumours only two types calcify, namely, the neuroblastoma and the adenocarcinoma of the cortex. In the former the calcification is speckled and poorly defined. It is seldom great in degree. The extent of calcification in an adenocarcinoma is proportionate to the extent of hæmorrhage into the tumour. It may be poorly defined, as in a neuroblastoma, or massive. The frequency of calcification in adenocarcinoma of the adrenal cortex is difficult to estimate owing to the relative infrequency of the condition. The association of a calcified adrenal tumour with the clinical manifestations of the adreno-cortical syndrome is diagnostic of a carcinoma of the adrenal cortex.

PHYSICAL THERAPY.

Naso-Pharyngeal Carcinoma.

V. P. GRAHAM AND RALPH MEYER (*Radiology*, January, 1948) state that malignant tumours of the naso-pharynx are rare, and as the tumour in the early stages is difficult of access, many are missed. More than half of the patients in this series had had operations for relief of symptoms without recognition of the primary lesion. The series is of 26 patients treated by X-ray therapy. The chief complaint in 16 cases was a cervical mass, in six cases pain; 18 of the 26 patients had palpable lymph glands and 15 had involvement of one or more cranial nerves; 17 showed varying degrees of destruction of bony structures at the base of the skull. The authors comment that primary symptoms produce few early signs, which accounts for the advanced stage at which most of the patients were seen. A biopsy was carried out on every patient and seventeen were found to have transitional cell carcinoma. The method of treatment aimed at delivering a dose of 5000*r* to the tumours through three ports. Only six of the patients are now living, five of whom have survived over five years; one patient died from recurrence seven years after treatment. The average survival time of the patients who died was twenty-three months.

Artificially Prepared Radioactive Isotopes.

EDWARD REINHARD (*American Journal of Roentgenology*, December, 1947) describes nuclear changes involved in the production of radioactive phosphorus. The β ray which is emitted from P^{32} penetrates at a maximum 0·7 centimetre into the body tissues. Experiments on mice and post-mortem examination of patients treated by radioactive phosphorus show that radioactive phosphorus is used in the synthesis of nucleoproteins and accumulates in largest amounts in these tissues which are primarily involved in *polycythæmia vera*, the leuchæmias and lymph node disease. The effect of treatment is as follows. In *polycythæmia vera* excellent clinical and hæmatological remissions can be obtained in the majority of cases. The erythrocyte count never shows a significant decrease with therapeutic doses in less than three weeks, and it may be three months before the red blood cell count is stabilized at its lower level. The recommended course of treatment is six to eight millicuries given over three or four weeks, and, as stated, the full effect may not be obtained for three months. The average interval before a second course is called for by return of symptoms is about thirteen months. The author has not been able to deduce whether the expectation of life is prolonged as the patients have been followed for too short a time. All investigators have agreed that P^{32} is of little or no value in the treatment of acute myeloid leuchæmia. In chronic myeloid leuchæmia, radioactive phosphorus produces as complete clinical and hæmatological remissions as deep X-ray therapy and has the advantage of not producing irradiation sickness. It prolongs life no more than deep X-ray therapy. In chronic lymphatic leuchæmia, radioactive phosphorus produces less satisfactory symptomatic relief and reduction in size of the lymph nodes. It appears to be less effective than deep X-ray therapy in this disease. In no other condition in which it was tried did P^{32} prove of any value. Most of the preparations of radioactive iodine consist of a mixture of I^{130} and I^{131}. Both emit β and γ rays. The normal thyreoid accumulates eighty times the general body cell amount of iodine and the hyperplastic thyreoid up to several hundred times the amount. Herz and Roberts have a series of 28 patients with hyperthyreoidism treated with radioactive iodine given orally. Five patients subsequently underwent thyreoidectomy. Of the remaining 23, 20 were no longer thyreotoxic after three to five years. In carcinoma of the thyreoid, the malignant cells lose their ability to take up iodine and only a rare case will respond to this form of treatment. The radioactive isotopes of calcium and strontium have given disappointing results and this type of therapy has been abandoned. Radioactive sodium Na^{24} has no selective uptake by bone or bone marrow, but chronic leuchæmia treated by this responds favourably, just as it does to general body irradiation. The author concludes that the radioactive isotopes of inorganic elements have only a limited field in treatment, but work is in progress on selective concentration in cancer cells of organic compounds which may yield better results if these can be synthesized from radioactive elements.

Carcinoma of the Bronchus.

L. M. SHORVON (*The British Journal of Radiology*, November, 1947) discusses the increase in the incidence of carcinoma of the bronchus, various theories of its ætiology and the pathological types and symptoms. With regard to the treatment of the condition it is pointed out that surgery, when practicable, is the best method of treatment giving a reasonable percentage of five-year cures. However, it is shown that when patients are first seen only about 40% of the lesions are clinically operable, and thoracotomy on these patients will reveal that less than half that number are suitable for pneumonectomy. In most series then, less than 20% of the patients are suitable for operation. As far as treatment by radiotherapy is concerned, there is a wide difference of opinion concerning its efficacy, from those who consider it has no value, even as a palliative measure, to Leddy and Moersch, who consider that irradiation can produce cures. The latter authors had a series of 250 cases of proved bronchogenic carcinoma treated by deep X-ray therapy. Twenty-five of these patients survived from one to eleven years. The views of numerous other workers with less encouraging results are also given. The author stresses the fact that before treatment is given it must be decided whether treatment is to be radical or palliative. Indications for palliative treatment are good general condition of the patient and the ability to localize accurately the tumour area. The various techniques of treatment are discussed, including the methods of accurate beam direction. The author has been giving penicillin 250,000 units intramuscularly twice a day during treatment and considers that this has lessened pyrexial attacks and helped to maintain the patient's condition. The present series of 213 patients were treated at Mount Vernon Hospital between 1942 and 1946; 75 of these were too ill for deep X-ray treatment, 23 had palliative treatment and 111 radical treatment. No patient treated has so far survived for more than three years, but of the 111 patients treated radically 28 are still alive. The results are not impressive, but, as Shorvon points out, many of these patients had great relief of such symptoms as pain and dyspnœa and some were able to go back to work for variable periods. The average time of survival was fifteen and a half months from the onset of symptoms as compared with about nine months among similar untreated patients.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on December 4, 1947, at the Saint George Hospital, Kogarah. The meeting took the form of a number of clinical demonstrations by the members of the honorary staff of the hospital. Part of this report appeared in the issue of July 3, 1948.

Chronic Neutropenia.

DR. J. C. ENGLISH showed an albino woman, aged thirty-nine years, suffering from chronic neutropenia. The patient had always been well and had led an active life. On April 28, 1946, she had been admitted to the Saint George Hospital with a diagnosis of pelvic cellulitis. A routine leucocyte count had revealed leucopenia with a neutrophile cell count of only 6%. This had continued despite treatment with penicillin, "Pentnucleotide", blood transfusion, liver extract *et cetera*, until her discharge from hospital, apparently well, on June 22, 1946. No drugs could be implicated as causing the neutropenia. She was readmitted on August 5, 1946, with a history of coryza for three days, of cough, rigors and vomiting present for one day. There were signs of patchy consolidation over the lower lobes of both lungs. Her temperature ranged up to 105° F. The leucocyte count on the day after her admission to hospital was 6800 per cubic millimetre, of which 3600 were neutrophile cells. After eight days the temperature had fallen to normal by lysis and the neutrophile cell count to 600 per cubic millimetre. The patient's condition was much improved. During the next two and a half weeks the temperature continued to be normal and the neutrophile cell count remained below 500 per cubic millimetre. X-ray examination on August 20, 1946, revealed an area of pneumonitis in the lower lobe of the left lung and signs still persisted there on August 29. On August 31 and September 1 there was a brief febrile period followed by a rise in the neutrophile cell count to 740 per cubic millimetre on September 2. X-ray examination on September 3 revealed a lessening in the extent of the inflammatory area in the lower lobe of the left lung. On September 16, 17 and 18 there was another febrile period with pain in the left side of the chest and diminished breath sounds.

The patient's condition gradually improved during the subsequent week. The temperature again rose on September 24 and 25. On September 30 X-ray examination showed that the base of the left lung was normal, but there was a new area of pneumonitis in the mid-zone of the same lung. On October 1, 2 and 3 the temperature again rose and pain was again felt in the left side of the chest. No *Mycobacterium tuberculosis* could be found in the sputum. On October 7 pyrexia recommenced and continued until the time of the clinical meeting as a continuous fever ranging between 99° and 104° F. Sternal biopsy showed interference with maturation and liberation of granular cells. There was some increase in the number of lymphocytes. On October 20 the Widal reaction could not be elicited and there was no agglutination with *Bacillus paratyphosus* A or B, with *Brucella abortus* or with *Bacillus proteus* OXK and X19. On October 21 a skiagram showed an increase in the extent of the mid-zone consolidation in the left lung. Examination of the sputum again failed to reveal *Mycobacterium tuberculosis* on October 29 and the result of the Wassermann test was negative. On November 5 cold agglutinins were present in the patient's serum to a titre of 1 in 32. No organisms were grown from blood taken on November 7. On November 21 cold agglutinins were present to a titre of 1 in 8, and the cerebro-spinal fluid was normal. During this time the neutrophile cell count continued to be low, rising to a maximum of 500 to 2000 per cubic millimetre after periods of pyrexia. The last count on November 29 was 2000 neutrophile cells per cubic millimetre. Penicillin was given in dosage of 15,000 units three hourly from August 12 to 25, from September 25 to October 25 and from November 10 onwards. "Pentnucleotide" was given from August 18 to 30. Folic acid was exhibited for a period of two weeks. Full dosage of vitamins, injections of liver extract and repeated small blood transfusions were administered without effect on the neutrophile cell count and on the course of the disease. At no time was the spleen palpable. The platelet count was always within normal limits. The patient at the time of the meeting was rapidly deteriorating, signs in her chest were extending, and a diagnosis of her condition had not been made. The combination of neutropenia without significant organisms in the sputum and cold agglutinins in a patient who did not look distressed had suggested a primary atypical pneumonia, but the length of history and serious deterioration of the patient's condition had again left the diagnosis open.[1]

Crohn's Disease.

DR. G. F. ELLIOT discussed the clinical history of a female patient, aged seventy-three years, who on January 26, 1946, had undergone an operation for removal of a pre-carcinomatous *cervix uteri* and cure of severe *prolapsus uteri*; she had used a combined watch spring pessary for six years. She was discharged from hospital on March 12. On return to her home she had occasional bouts of vomiting, sometimes copious, but not accompanied by pain. These attacks gradually subsided and she seemed well for a time. Later she again began to vomit. There was still no pain, but anorexia was pronounced even between bouts. Her general condition began to deteriorate, constipation was troublesome and vomiting was almost continuous. Apart from some distension there were no findings on abdominal or rectal examination. She had consulted a doctor only once in her life, for a cervical erosion.

X-ray examination on April 27 revealed obstruction in the jejunum just beyond the duodeno-jejunal flexure with considerable duodenal stasis apparently occasioning gastric delay. After four hours only a small amount of barium had passed into the jejunum distal to the site of narrowing. She was admitted to hospital for operation with a provisional diagnosis of carcinoma of the jejunum.

At operation on May 1 the first part of the jejunum a few inches beyond the flexure was found somewhat enlarged and thickened, with grossly thickened and indurated mesentery. The condition did not resemble carcinoma in appearance or to the touch. About fifteen inches of the jejunum were removed and end-to-end anastomosis was performed with considerable difficulty on account of the short and fixed proximal segment of the jejunum. On the patient's return to the ward Wangensteen suction was instituted and she was given fluids intravenously. These were discontinued on May 3, as she was well and taking fluids freely by mouth. On May 4 she was able to attend to her own toilet, but slight swelling of the left leg was noticed. At about 10 p.m. on May 5, after requesting a bed pan, she collapsed and died almost immediately. A provisional diagnosis of pulmonary embolism was made.

Dr. N. Powys noted the following findings on the operation sheet:

> Almost complete obstruction 2-3 inches beyond duodeno-jejunal flexure and beyond for 10 inches is a lesion in which there are lumps in the wall of the bowel, rugæ of the bowel lost and mucosa showing some atrophy.

Autopsy was performed on May 6 by Dr. C. S. Graham. Examination of the heart revealed an embolus in the pulmonary artery bifurcation; most of the embolus had lodged in the right pulmonary artery, but some was also present in the left. Old pleuritic adhesions were present at the base of the right lung (possibly the site of a former embolus). In the rest of the heart and lungs no abnormality was detected. Examination of the abdomen showed the wound and operation area to be healing satisfactorily; the anastomosis was apparently functioning well. The remainder of the bowel was normal. Thrombosis was present in the left femoral vein and extended as high as the junction of the common iliac veins, where the clot had broken off. Examination of the saphenous vein revealed no thrombosis in the femoral triangle. Old thrombosis was present in the left renal vein. A pale old infarct was found in the middle third of the left kidney. In the right kidney chronic interstitial nephritis was present with increased pelvic fat and narrowing of the cortex. Both capsules stripped with difficulty. The liver was normal except for a large solitary cyst of the right lobe on the diaphragmatic surface. The spleen was normal.

Dr. A. E. Gatenby's pathological examination revealed the following findings:

> Sections show ulceration of the mucosa with replacement of the mucosa in the ulcerated areas with fibrinous

[1] This patient died shortly after the clinical meeting and post-mortem examination revealed extensive miliary tubercular infiltration of lungs, liver and spleen. Numerous tubercle bacilli were to be seen in sections of these organs stained by the Ziehl-Neelsen method. The left lung was consolidated throughout the whole of the lower lobe and two-thirds of the upper lobe. There was a small necrotic cavity in the upper lobe of the left lung. There was a small consolidated area in the middle lobe of the right lung. Old scarring and adhesions were present at both apices.

material containing inflammatory cells of the polymorphonuclear, lymphocyte and endothelioid cell type.

The intestinal wall is thickened and shows a diffuse infiltration extending through to the serous coat with chronic inflammatory cells mainly of the plasma cell, lymphocyte, and endothelioid cell type.

Several large multinucleated cells, along with focal accumulations of the inflammatory cells, are present. No lymphoid follicles can be recognized, but in some areas the arrangement of the inflammatory cell groups suggests the destruction of the normal lymphoid follicles.

The appearances are those of a chronic regional enteritis analogous with the regional ileitis as described by Crohn.

Dr. Elliott's next patient was a male, aged sixty-six years, who had been admitted to hospital on July 15, 1947, with a diagnosis of strangulated femoral hernia with auricular fibrillation and coronary occlusion. On the previous night he had had a sudden attack of precordial pain, dyspnœa and coughing. Later, during a bout of coughing, he experienced sudden pain in the centre of the abdomen, and a right-sided hernia, known to exist for some months previously, came down and was irreducible. Subsequently he vomited several times, but had a normal bowel action.

On examination of the patient the pulse was irregular in time and amplitude. The heart sounds were of poor quality and irregular. Dulness was present at both lung bases with numerous râles and crepitations. A large, hard, irreducible mass was present in the right groin; it was not tender on palpation. The abdomen was distended and diffusely tender, but not rigid. The liver edge was palpable one finger's breadth below the costal margin. A provisional diagnosis was made of strangulated femoral hernia and auricular fibrillation due to a coronary occlusion (this was later confirmed by an electrocardiogram).

At operation, under local anæsthesia and cyclopropane anæsthesia, a strangulated femoral hernia was found containing a loop of small bowel, black and almost lustreless. The sac was excised. In view of the patient's general condition, it was decided merely to replace the bowel. He was given "Digoxin". Convalescence, apart from a mild inflammatory reaction in the wound, was uneventful, and he was discharged from hospital on August 24.

When readmitted to hospital on September 16, 1947, the patient said that he had not been well since his discharge three weeks earlier. During the first week he had had attacks of colicky abdominal pain with borborygmi, had vomited twice and had had slight diarrhœa. During the week preceding his readmission he complained again of intermittent abdominal pain, but had regular daily bowel motions. An attack of pain worse than before began on September 16 and his bowel did not act. The pain began in the epigastrium and spread all over the abdomen, then passed away in spasms. He vomited "dirty fluid" four times. There were no symptoms suggestive of further cardiac damage.

On examination of the patient his abdomen was distended, resonant and not tender, and contained no palpable masses. His tongue was moist, but somewhat dirty, and his breath was offensive. His pulse rate was 60 per minute, and his blood pressure was 140 millimetres of mercury (systolic) and 100 millimetres (diastolic). An enema was given with a good result and relief of symptoms. He was radiologically examined by means of an opaque enema on September 19, and the following report was submitted: "The enema ran from the rectum to the caecum and through the ileo-caecal valve into the terminal ileum without revealing any abnormality." However, as it was thought on clinical grounds that obstruction was present in the small bowel, an opaque meal and follow-through examination was requested, and on September 25 a report suggesting a partially obstructing lesion in the small bowel was obtained. At operation on October 1 under anæsthesia with nitrous oxide, oxygen and ether with curare, a mass of small bowel which at first sight suggested Crohn's disease was found adherent to the parietal peritoneum of the right iliac fossa and over the opening of the femoral canal. A peritoneal band constricting a loop of small bowel was ligated and divided. Separation of the coils of bowel adherent to the peritoneum was undertaken—a matter of considerable difficulty—and when the task was almost complete the bowel was perforated. About eight or nine inches of diseased bowel were resected and lateral anastomosis was performed. The specimen was sent for pathological examination as a possible example of Crohn's disease. Convalescence was disturbed by a persistent cough with much tenacious sputum. This caused the bursting of a tension suture, and it was feared that eventration would occur. However, there were no further developments and the patient was discharged from hospital on October 28.

Dr. Gatenby's pathological examination produced the following findings:

The specimen was 7″ of excised small intestine. About the middle of the specimen the wall of the bowel was discoloured and thickened. Sections were taken from this area.

Microscopic. Sections show areas of destruction of the mucosa and a diffuse infiltration of the mucosal surface and intestinal wall with inflammatory cells mainly of the polymorphonuclear and plasma cell type. The appearances are those of a subacute regional enteritis and could be considered as analogous with an early stage of the regional ileitis described by Crohn. No evidence of malignancy was seen.

Intestinal Obstruction: Ileum in Retrocaecal Pocket.

Dr. Elliott finally showed a male patient, aged twenty-one years, who had undergone an appendicectomy for acute appendicitis on July 18, 1945, and had been discharged from hospital on July 28. He was readmitted to hospital on June 27, 1947, with a history of abdominal pain, hiccup and nausea, but no vomiting; the last bowel action had occurred two days previously, and an enema had been given without result on the day of his admission to hospital. He was operated upon immediately with a provisional diagnosis of intestinal obstruction due to a peritoneal band. At operation the following condition was actually found. A loop of ileum had become incarcerated and obstructed in a retrocaecal pouch, from which it was liberated with some difficulty on account of adhesions. An attempt was made to close the opening to the fossa by suture. Some vomiting occurred on the second and third post-operative days. On the seventh post-operative day the patient complained of some pain in the right side of his chest, and some crepitations were found at the base of the right lung. This condition subsided quickly on the administration of penicillin, and he was discharged from hospital on July 18.

(To be continued.)

Special Correspondence.

LONDON LETTER.

FROM OUR SPECIAL REPRESENTATIVE.

"They that Rule . . . in Stately Conclave Met."

THE Annual Representative Meeting of the British Medical Association was held at Cambridge this year and began on Friday, June 25. There is a special atmosphere about meetings in university towns, as it means the renewal of old associations to many of the representatives. Thus the writer was taken to lunch by the editor of the *British Medical Journal* at a café which had been opened when the latter was a student and which is still under the same management. This was followed by a personally conducted tour over the host's old college, Trinity, made particularly interesting by the guide's fund of information and stories. A well-known handbook states that "Cambridge in vacation just lives quietly from one market day to the next". The Annual Representative Meeting met in vacation time, but even then the narrow streets, many of them "one way only", seemed anything but quiet. It is said that at Saint Andrews everyone talks and plays golf; at Cambridge everyone seems to own and ride a bicycle; the resulting crush when the university is "up" must be bewildering. The colleges generously opened their doors to the visitors and your representative was allocated to Newnham, a college for women students, where, owing to holidays and the consequent shortage of staff, making your own bed was the order of the day. It was rumoured that two eminent doctors staged a competition in this connexion, their wives to act as judges. So far no results have been published, but the adjudicators are said to have been disappointed at the lack of skill shown in such a simple task; perhaps the contestants were wise in this respect. A college built for use by women has certain disadvantages to the male intruder in that there is an absence of mirrors in bathrooms and places where men shave. The old soldier, trained to prepare for the worst and to hope for the best, brought his mirror with him, as did the antifeminist; others less provident had to descend to

various devices, each according to his own ingenuity. Apart from this structural drawback, the stay in the unaccustomed environment was most pleasant and the garden was a constant joy. What the college authorities thought of the intrusion is not known, but, if the verdict was unfavourable, it was admirably concealed.

The business dealt with by the representatives was for the main part domestic; some reference was made to the immediate past, but on the whole the discussions were good-tempered, informative, well argued, and looked towards the future. Sir Hugh Lett, who has been a stalwart upholder of a closer liaison with overseas branches during his time as president, was succeeded by Sir Lionel Whitby, and; from personal knowledge of the latter, it is safe to assert that the same policy will be followed. The new president is perhaps best known for his work on the "sulpha" drugs in pneumonia and as the head of the Blood Transfusion Service during the recent war, which was brought to a high state of efficiency under his direction. Dr. J. B. Miller, the retiring chairman, was in good form. While the standing orders, under which the meeting works, were being discussed, the usual motion to suspend the one prohibiting smoking was moved. A meticulously minded representative suggested that it was out of order to suspend one particular standing order before the standing orders as a whole had been approved. "J.B." got out of this dilemma by ruling that "provisional smoking would be allowed".

A full report of the proceedings will appear in the *British Medical Journal*, but the following notes may be of interest. The first serious discussion arose over a demand that a committee be set up to investigate the happenings which had led Council to call for the April plebiscite. It was argued that as the policy of the Socialist Government for a full-time salaried service still stands, the whole question should be gone into and a report drawn up which might be of help in the future, should any similar position again arise. "The profession and the public are mystified, and if there is nothing to hide no harm can be done by an inquiry." "Such an inquiry would refute idle criticism." This was opposed by several speakers, including Dr. Dain, Chairman of Council, and the motion was lost on a show of hands. The representatives coming from outside London and other big cities always feel at a disadvantage at the voting power carried by these dense centres of population, medical and otherwise. To counteract this a system of block voting has gradually grown up. Representatives of an area agree on a nomination for a post and by canvassing and not splitting the vote do their best to get him in. It is a case of regions rather than personalities. A motion to debar this practice was lost only on a count. Much attention was directed to the various Spens reports on remuneration and the Council was instructed to press for full implementation of the recommendations set out therein. Pressure was exerted to have an increased and more equitable mileage allowance; country representatives stressed the disadvantage they were under in this respect. They also pointed out that a man with a scattered practice could not accept as many patients on his list as a practitioner in an urban area, and suggested that the capitation fee should be on a sliding scale to overcome this difficulty. A similar claim was urged on behalf of the coastal and other health resorts, where, owing to the greater preponderance of ailing and aged inhabitants, more visits *per annum* would be necessary than in an ordinary area where most of the people on a doctor's list were younger and in better health.

After July 5 the consultants and specialists will come into line with the general practitioners in that they will require a body to conduct negotiations with the Ministry of Health on such matters as terms of service, method of appointment, remuneration and the like. In the past, work of this nature has been done for the general practitioners by and through the Insurance Acts Committee of the British Medical Association. In matters of an academic nature the consultants look to the Royal Colleges, and to meet the new need a British Medical Association Committee has drawn up a comprehensive scheme which provides for the formation of Regional Consultant and Specialist Committees and the establishment of a Central Consultants and Specialists Standing Committee; this organization was approved by the Representative Body. It was further decided that the Central Standing Committee should be an autonomous body with full power to determine policy in its own sphere, such decisions not to be subject to approval by the Council of the British Medical Association or by the Representative Body as is the case with other standing committees; the decisions would, however, be implemented through the administrative machinery of the Association. The Central Committee would elect its own chairman, and to maintain liaison he would *ipso facto* become a member of the British Medical Association Council. The British Medical Association feels that it has the central machinery and peripheral organization to make such a Central Committee a real power, whereas the Royal Colleges have not; already 11 out of the 14 regions established under the acts have set up the necessary Regional Committees. The reactions of the Royal Colleges to this arrangement, which has gone down well in the provinces, remain to be seen. The President of the Royal College of Surgeons evidently supports it, for he writes: "The Royal College of Surgeons is an academic body. . . . I quite agree that the College is not so constituted as to be an adequate body for day to day medico-political negotiations." (*British Medical Journal*, July 3, 1948, page 46.) A suggestion to press for a forty-hour week for doctors was turned down quickly and decisively, but the Council was instructed to explore the possibility of forming an organization, along parallel lines to the British Medical Association, but having certain of the advantages which belong to a trades union, notably as to the powers, to safeguard the working conditions and pay of its members.

Members of Council and of the Representative Body are paid first-class return railway fares, and sleeper fares if necessary, for all meetings they attend. The question of additional payment for subsistence is a hardy annual and came up as usual, but was once more turned down. In addition to the usual reports from the standing committees consideration was given to one on health centres under the *National Health Services Acts* and another on the *Coroners' Acts*. The report on health centres is a most comprehensive document and was referred to Council for consideration. Much study had been devoted to the *Coroners' Acts* and stress was laid on the need for the practitioner in previous attendance being given every facility to attend any post-mortem-examination or to receive a copy of the report. The meeting also approved a recommendation that "in the case of an inquest on a 'suicide' the Press . . . be permitted only to publish the fact that an inquest had been held, the name and address of the deceased, and a verdict that the deceased died by his own hand". Other recommendations concerned qualifications for coroners, scale of fees for post-mortem examinations and the conducting of necropsies by competent men with the aid of proper facilities and equipment. The usual elections were carried out during the meeting, and the most notable changes were Dr. E. A. Gregg moving up from Deputy Chairman to Chairman of the Representative Body in place of Dr. J. B. Miller, and Mr. A. M. A. Moore replacing Dr. J. W. Bone as Treasurer. The Council showed its appreciation of Dr. Bone's service by awarding him the Gold Medal, the highest honour in the gift of the Association. The representatives expressed their thanks to the retiring officers by standing up and applauding vigorously. The meeting ended dead on time, 12.30 p.m., Tuesday, June 29.

"A . . . Friend is the Medicine of Life."

At a luncheon given to overseas representatives, Sir Hugh Lett outlined some of the steps taken to implement the desire for closer cooperation between the parent body and medical men of the Empire. The British Commonwealth Medical Council is to have its first meeting in September, at which it is hoped all the Dominions will be represented; the two Australian delegates can be sure of a warm welcome. While the British Commonwealth Medical Council will deal mainly with matters of policy, the Empire Medical Advisory Bureau, now in being at British Medical Association House with Dr. H. A. Sandiford as director, will be of more interest to individual doctors visiting the United Kingdom. The Bureau will advise not only on post-graduate facilities, but also on all the general matters which a newcomer meets with during his stay. The arranging of social contacts and hospitality will be a feature of the Bureau's work. In support of this Dr. Sandiford gave a very pleasant cocktail party at Downing College which was well attended by overseas men and women. A booklet setting out the aims of the Bureau is in preparation and copies will be sent to Branches abroad. Dr. Sandiford should prove an ideal director, as during his military career he travelled widely and so is aware of Empire conditions. Branches overseas cannot be grateful enough to Sir Hugh Lett for the never-failing interest he has taken in Empire liaison during his term of office as president of the British Medical Association. The representatives of overseas constituencies were officially welcomed by the Representative Body at its meeting on Monday, June 28. Those from the Colonies painted rather a grim picture of the terms of appointment and conditions under which they work. Dr. Wright (Kenya) foretold a severe famine in his area within the next fifteen years, owing to the increase in the native population, unless tuberculosis and syphilis redressed the balance. Greetings

were brought by Dr. G. G. L. Stening (New South Wales), Dr. Gowland (Victoria) and Dr. Lumb (South Australia); all spoke briefly and well, and referred to the difficulties Australia may have to face in connexion with her *National Health Service Act*. Reference was also made to "Free Medicine", in connexion with which Dr. Gowland said the profession was "actively engaged in non-cooperation".

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

WEEK-END COURSE AT BROKEN HILL.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a post-graduate week-end course will be held at the Broken Hill and District Hospital, in conjunction with the Broken Hill Medical Association, on Saturday and Sunday, July 31 and August 1, 1948. The programme will be as follows:

Saturday, July 31: 2 p.m., registration; 2.30 p.m., "Problems of Fluid and Electrolyte Disturbance in Pædiatric Practice", Dr. Lorimer Dods; 4 p.m., "The Obstetric Pelvis and its Measurements", Dr. D. G. Maitland.

Sunday, August 1: 10 a.m., "Recent Progress in Pædiatrics", Dr. Lorimer Dods; 11.30 a.m., "Intestinal Obstruction", Dr. Norman Wyndham; 2.30 p.m., "Opaque Meals", Dr. D. G. Maitland; 4.30 p.m., "Surgery of Peptic Ulcer", Dr. Norman Wyndham.

The fee for attendance will be £2 2s., and those wishing to attend are requested to notify Dr. F. Schlink, Honorary Secretary, Broken Hill Medical Association, Broken Hill, as soon as possible.

Correspondence.

MEETING OF THE AMERICAN PSYCHO-ANALYTICAL ASSOCIATION.

SIR: The annual meeting of the American Psycho-Analytical Association was held here on May 15 and 16. The members were divided into six groups, each group devoting the whole of one day to a very full discussion of its own particular problem. There were six such groups: (i) study of social issues, (ii) therapy and treatment of schizophrenia, (iii) indications and criteria for child analysis, (iv) problems of psycho-analytical training, (v) psycho-analyses and psychotherapy, (vi) psycho-analysis of the creative imagination. Some four hundred members took part in the discussions. These numbers give some indication of the marvellous growth of psycho-analysis in the United States of America. Many of the most notable psychiatrists of the nation were present and took part in the discussions.

I was not able to attend more than one group. I chose the group therapy and treatment of schizophrenia. Dr. Fromm Reichman introduced the subject by indicating the psycho-analytical conception of schizophrenia and gave some results of her treatment by prolonged and very careful analysis; she claimed some 50% of cures. Dr. Louisa Despert, of Baltimore, spoke of studies upon children which showed that at three years of age the normal child could distinguish between phantasy and reality, and at this same age the analyst could differentiate the normal from the schizophrenic child. The highlight of the discussion was Dr. John Rosen, of New York. Dr. Rosen was practising pathology until about 1943, when he gave it up and entered a State mental hospital and studied psycho-analysis in relation to psychiatry. He became disgusted with the irrational treatments of psychoses, shock therapies *et cetera* and went into private practice determined to attempt the cure of schizophrenia. Before leaving the hospital he made an attempt by means of direct forceful interpretation upon a chronic catatonic. To his surprise he cured this incurable patient. He continued to practise direct analytical treatment upon a series of schizophrenics ranging in age from fourteen to fifty-three years. His interviews varied from one hour up to eight hours' continuous treatment varying from once or twice a week to five times a week. He claims to have cured 52 out of 53 severe schizophrenics. There can be no doubt about his cures and those of Dr. Fromm Reichman. They were abundantly vouched for by others. No one, not even Dr. Rosen, claims to know just how his therapy works, but today the eyes of the American psychiatric world are upon Rosen. He has accomplished the seeming impossible. I have met the man; he greatly impressed me. He is physically short in stature, powerfully built, and has a most pleasing manner and is devoid of fear and anxiety. If Dr. Rosen's claims are substantiated by other workers it can be said that he has made the greatest advance in psychotherapy since Freud.

Following immediately upon this conference there was held the one hundred and fourth annual meeting of the American Psychiatric Association. Some two thousand five hundred members from all over the United States of America assembled. There were a few from Great Britain and Europe and Canada and myself from Australia. This huge congregation of men included not only psychiatrists but philosophers and theologians and social workers. Most of the psycho-analysts stayed on for this conference, and the first afternoon was devoted to a combined meeting at which a very learned discussion upon "Faith and Psychotherapy" under the chairmanship of Dr. Gregory Zilborg took place.

The conference was divided up into four main sections; each section met morning and afternoon for four days and in the evenings round-table conferences were held during supper. Here discussion was informal and intimate and more valuable than the mere reading of papers. All the hundred and one aspects of psychiatry were discussed from faith to such laboratory studies as the effects of electroconvulsive therapy upon water metabolism. The old controversy as to whether schizophrenia is organic or functional or a combination of the two was not allowed to die, as two pathologists gave a paper on the histopathology of schizophrenia, in which they expressed the opinion that as a result of their studies they regarded this disorder as among the organic psychoses. Dr. Lewis Goodman described 233 cases of Alzheimer's disease, the clinical features being analysed and correlated with the pathological findings.

The two discussions which aroused the greatest interest were those on prefrontal lobotomy and group psychotherapy. Three papers on lobotomy were read, one of these being by Dr. Walter Freeman; it was lavishly illustrated by lantern slides of patients before and after operations, of the anatomy of the operation *et cetera*. Dr. Freeman is, of course, a neuroanatomist of renown. He has now become a neurosurgeon, at any rate as far as his operation of lobotomy is concerned. The patient is anæsthetized by electro-shock; then a knife is inserted under the upper eyelid and pushed up through the orbital plate, and a limited area of the anterior and medial portion of the frontal lobe is severed. Dr. Freeman claims that patients operated upon by this method are up and out of bed the next day and exhibit none of the post-operative problems such as enuresis, fits *et cetera*, that follow the more orthodox operation of Dr. Watts. Further, few if any undesirable behaviour problems ensue. There appeared to be a general opinion that prefrontal lobotomy benefits a certain number of chronic schizophrenics who exhibit impulsive, excited, destructive behaviour patterns. It also benefits a large number of chronic agitated involutional melancholics. I was not able to find the same enthusiasm for Dr. Freeman's work that he apparently does, and others to whom I spoke were in agreement with me.

The discussion on group psychotherapy was very interesting and informative. There was a general consensus of opinion that we have in group therapy an extremely helpful and additional means of treating large numbers of sick people. Not only those sick of neurosis, but also the psychoses and problem children. Probably the most striking aspect of group therapy is that by this method the relatives of the patient can also be treated. In this regard we have a therapeutic means that has long been the desire and hope of psychiatrists. We know that relatives are often sick like the patient, and in fact are a cause of the patient's ill health and maladjustment. I was tremendously impressed by the procedures adopted in group therapy and about the possibility of psychiatric help that could result from such an approach.

Moving pictures were shown of the behaviour of diverse types of babies to frustration. There were also films that depicted the growth of neurotic patterns from early childhood up to adult life, and also films of the series of events in the lives of soldiers that antedated and accompanied the growth of various types of neuroses in war. Another film showed the changes following prefrontal lobotomy, the actual patient having been filmed before and after operation. These

films showed almost unbelievable changes in the facial expressions from those of very deteriorated psychotics to those of a normal person.

Another very striking exhibition was that shown by ex-soldiers. This consisted of about twenty scenes arranged as on the stages of theatres depicting the treatment of psychoses down the ages. Prehistoric man was the first; here we see the unfortunate lunatic being cast out of the horde into the wilderness; then we see the various methods, chains, stocks, holes being bored in the skull, roasting the head in an oven *et cetera*, which were common in the Middle Ages. The modern age is ushered with a scene showing the noble Pinel knocking off the chains in the Bicet. After this comes Freud lecturing to his first students all gowned in white robes, and lastly are shown the scenes of modern hospitals, shock therapy, psychotherapy, dancing, music *et cetera*. This was a marvellous pictorial history.

These learned psychiatrists, however, proved themselves to be human, all too human. They concluded the scientific deliberations with parties and banquets at which they offered up libations in the many forms of beautiful alcoholic drinks which are so plentiful and so fascinating in this amazing America.

Yours, etc.,

PAUL G. DANE.

Washington, D.C.,
United States of America,
May 19, 1948.

PROGNOSIS IN BREAST CANCER.

SIR: In your journal of June 5, 1948, you publish a letter from Mr. Robert Fowler on prognosis in breast cancer which concludes with the following paragraph:

> As regards the effect of post-operative irradiation on prognosis, the Melbourne sample may be divided into a subgroup of 148 cases given post-operative X-ray therapy and a second subgroup of 99 cases not so treated. The respective five-year survival rates prove to be 46·6% in the former and 46·5% in the latter.

As Mr. Fowler claims, the Melbourne cases may be divided in this way, but I suggest that it is quite unprofitable, and perhaps actively misleading, to do so without the submission of further information. It may be assumed that the "surgery" considered in this table is a radical removal of the breast, muscle and glands carried out by a trained surgical specialist. The radiotherapy, on the other hand, is far from standardized and will certainly vary in quality from hospital to hospital. With our state of knowledge of X-ray therapy it is not practicable to say which particular technique of application of the rays is the best, and it must be assumed that all are equally competent at present, but in future the effect of the technique of radiotherapy on prognosis will be comparable with, say, the effect of simple as opposed to radical mastectomy.

Equally important and more immediately available information is that concerning the reason for the choice of post-operative irradiation in some cases and its rejection in others. It may be that the matter is one of routine and that certain hospitals use post-operative X-ray treatment in all cases of breast cancer, while others never do. But this seems most unlikely, and it is probable that at least some surgeons exercise judgement as to which patients will be sent for X-ray treatment, with a result that the earlier stages of breast cancer will more often be found in the group treated by surgical means only, and the later stages in the group which has also received post-operative X-ray therapy. The danger of these figures then is that the casual reader may conclude from them that post-operative irradiation is of no value in this condition, whereas a more critical examination would show that the equality of figures represents a significantly increased salvage due to the X-ray therapy.

Yours, etc.,

B. S. HANSON.

163, North Terrace,
Adelaide,
June 23, 1948.

SIR: In the journal of June 5, 1948, there appeared a letter from Mr. Robert Fowler, giving certain statistics of survivals of patients suffering from cancer of the breast, prepared by the Cancer Registry of the Anti-Cancer Council of Victoria. The last paragraph of his letter reads:

> As regards the effect of post-operative irradiation on prognosis, the Melbourne sample may be divided into a subgroup of 148 cases given post-operative X-ray therapy and a second subgroup of 99 cases not so treated. The respective five-year survival rates prove to be 46·6% in the former and 46·5% in the latter.

I write this letter as I feel that these statistics, as presented, may be interpreted by readers as indicating that post-operative X-ray therapy is a useless form of treatment following radical mastectomy.

The figures, as presented, do not reveal all the relevant facts. Figures prepared by Lane-Claypon[1] in England show a post-operative survival for surgery alone of approximately 78% in Class I, and approximately 23% for Classes II and III combined. (Class I is defined as a growth still local, Class II as local carcinoma of the breast with involvement of axillary glands, and Class III as a growth extending to tissues outside the breast itself other than axillary glands; for example, skin fixation or ulceration or muscle fixation may be present, though the growth is still technically operable.) She examined a series of 2006 cases from representative English sources. Pfahler[2] produced almost identical survival figures following an examination of American statistics.

It is generally assumed that patients in Class I who succumb to cancer die entirely of distant metastases and are not subject to local recurrences. Under these circumstances no benefit is to be obtained from post-operative X-ray therapy of this group of patients, and Pfahler has shown that the survival rate is not appreciably altered by the addition of radiation therapy. X-ray therapy can have only a local effect, and a diminution in the incidence of local recurrences is to be expected. But no alteration can be anticipated in the incidence of distant metastases.

It is therefore only in the cases falling in Classes II and III that post-operative irradiation is regarded as essential by radiotherapists, and Pfahler has shown that the survival rate in these groups has been nearly doubled by adding radiation after operation. Five-year survival figures of 41·7% net have been obtained in my clinic at the Royal Melbourne Hospital for patients in Classes II and III with carcinomatous involvement of glands present at operation and treated subsequently with radiation therapy during 1937-1942. These results support Pfahler's experience.

The survival figures quoted by Mr. Fowler for surgery alone combine all operable cases, and so include patients in each of the three classes, I, II and III. It is improbable that the surgeon would send many of the early cases for post-operative irradiation, but more probably he would send those in which he doubted the completeness of the operation, or in which he saw evidence of advanced staging of the tumour. So the surgery only group might reasonably be expected to contain a greater proportion of the early classes, and the surgery *plus* radiation group a greater proportion of the cases in Classes II and III. If such a tendency to selection of cases does in fact exist, and this is, of course, but surmise, an unintentional fallacy in the comparison of figures offered by Mr. Fowler exists. Under the circumstances, further investigation should be undertaken to show that the groups are really comparable. Until this is done, conclusions from the figures he has given should not be drawn.

Further, it does not seem wise to group together three classes having prognosis rates which differ, as Lane-Claypon has shown, by figures as divergent as 78·5% in Class I and 21·9% in Class III. But Mr. Fowler unwittingly has done this, and so strikes an average of 46·5% in the surgery alone group for comparison with a surgery *plus* radiation group probably composed of different proportions of cases of Classes I, II and III.

An impartial examiner of the literature has great difficulty in finding the truth. Many obstacles are present, for no unanimity in staging methods exists, and uniformity of diagnostic standards is not attained. Until further statistical studies can be produced on comparable groups of patients it would be most wrong for the profession to accept the apparently obvious deduction from Mr. Fowler's figures, and thereby refrain from sending patients with operable carcinoma of the breast for the benefits of post-operative irradiation therapy. The radiotherapist will not insist on irradiating the proven Class I patients, but the weight of evidence which has so far been produced shows that in the later operable classes post-operative X-ray therapy is an essential accessory measure.

Yours, etc.,

R. KAYE SCOTT.

105, Collins Street,
Melbourne, C.1,
June 28, 1948.

References.

(1) J. E. Lane-Claypon: "Report on the Late Results of Operation for Cancer of the Breast", Ministry of Health Reports

on Public Health and Medical Subjects, Number 51, His Majesty's Stationery Office, London, 1928.

(3) G. E. Pfahler and G. P. Keefer: "The Object, the Value, and the Technique of Pre-operative and Post-operative X-ray Treatment in Carcinoma of the Breast", *Surgery, Gynecology and Obstetrics*, Volume LXXXV, July, 1947, page 35; G. E. Pfahler: "The Treatment of Carcinoma of the Breast", *American Journal of Roentgenology and Radium Therapy*, Volume XXXIX, January, 1938, page 1.

MEDICAL FEES AND NATIONALIZATION OF MEDICINE.

SIR: In view of the present danger of nationalization of medical practitioners I should like to point out certain facts which will militate against the adequate remuneration of medical practitioners in the event of nationalization becoming an accomplished fact.

It is certain that salaries of medical civil servants will be based upon present fees. I was reminded of this when seeing in Column 8 of *The Sydney Morning Herald* recently an account of a woman who sent for the doctor to treat her baby—fee 10s. 6d., and on the same date sent for a mechanic to inspect the refrigerator—fee 10s. It will be remembered by every one of my colleagues that if they get a plumber along to adjust the cistern of a lavatory, the amount charged, without considering any materials used, will be not less than the ordinary half-guinea fee charged by a medical practitioner when he visits the patient's home.

When I was a boy in the last decade of the last century the fee for the doctor coming to the house was exactly as it is today, 10s. 6d., but 7s. 6d. if the patient went to the doctor's surgery. When I first started practice in Macquarie Street in 1910 the fee was £1 1s. for each consultation, though a few people like Sir Alexander MacCormick and Sir Herbert Maitland charged £2 2s. for the first visit. At that time a consultant's fee in Harley Street was £3 3s. Within two or three years it became customary in Australia for the consultant to charge two guineas for the patient's first visit.

I think the only increase in fees has taken place in the matter of X-ray work, radiographic or radiotherapeutic. This increase has been forced upon medical practitioners by the vastly increased cost of apparatus, and especially of tubes and valves. But still the consultant's fee remains at £2 2s. for the first visit and £1 1s. for subsequent visits unless some special form of treatment has to be administered in the consultant's rooms or in private hospital. Up till the end of the 1914-1918 war these fees were paid in golden sovereigns, each of which then had nearly three times the purchasing power of the present pound note. To keep in step with this devaluation of the pound, medical fees should have gone up about 250%, but, as I have shown, this has not taken place.

It is well to ask ourselves what is to be done, especially in view of possible nationalization of medical practice and the salary to be regarded as a fair remuneration for medical services rendered, taking into account the greatly increased cost of living and the great increases in wages and salaries for non-medical services.

It is obvious, I think, that the medical profession have been very lenient in the matter of fees, and that consideration should be given to this matter in the near future.

I am informed by friends in the legal profession that legal fees have increased by 33%. The architect is the only professional man who has been adequately protected from the effect of devaluation of the pound, because his fees are fixed as a percentage of the total cost of the contract for building according to plans.

It seems the irony of fate that members of the medical profession, who alone among the professions (except the clerical) for a long series of years have done so much unpaid work for the community, both in hospital and private practice, should be singled out for nationalization as if they had been exploiting the public, whereas, as shown above, the reverse is obviously the case.

Moreover, if medical practitioners become civil servants, a forty-hour week and special terms for overtime will certainly be claimed. Therefore it will take two and possibly three civil servant medical officers to do what one private practitioner does today.

The average layman seems to think that a medical practitioner today is highly paid and can make a fortune out of his profession.

I can think of only one man who could be said to have made a rather modest fortune out of medical practice in my time. Some few others have become rich, but only as the result of business activities outside their profession.

I suggest that the time is now over-ripe for an increase in fees for medical services, if only to secure our colleagues from exploitation by the Government if or when the medical profession is nationalized.

Yours, etc.,

235, Macquarie Street, Sydney,
July 14, 1948.

E. H. MOLESWORTH.

The Royal Australasian College of Physicians.

THE following course of lecture-demonstrations at Prince Henry's Hospital, Melbourne, has been arranged for the months of August and September, 1948, by the Victorian State Committee of the College of Physicians.

Tuesday, August 3: "The Anatomy of the Bronchial Tree: Clinical Applications in Pulmonary Infections", Dr. G. Penington; demonstration of X rays, Dr. E. R. Crisp.
Thursday, August 5: "Recent Advances in Cardiology", Dr. K. Grice; demonstration of electrocardiograms, Dr. H. B. Kay.
Friday, August 6: Ward round, Dr. F. Blois Lawton.
Tuesday, August 10: "Diseases of the Gastro-Intestinal Tract", Dr. F. Niall; pathology demonstration.
Thursday, August 12: "Nephritis", Dr. M. Tallent; pathology demonstration, Dr. J. D. Hicks.
Friday, August 13: Ward round, Dr. H. H. Turnbull.
Tuesday, August 17: "Recent Advances in Blood Disorders", Dr. J. McLean; demonstration of blood films *et cetera*, Dr. G. Harkness.
Thursday, August 19: "Children's Diseases", Dr. S. Williams; pathology demonstration.
Friday, August 20: Ward round, Dr. K. Fairley.
Tuesday, August 24: "The Management of Diabetic Patients", Dr. T. A. F. Heale; pathology demonstration, Dr. J. D. Hicks.
Thursday, August 26: "The Management of Hypertension", Dr. J. L. Frew; demonstration of fundi, Dr. K. O'Day.
Friday, August 27: Ward round, Dr. M. D. Silberberg.
Tuesday, August 31: "The Principles of Diagnosis and the Use of Special Methods of Investigation", Dr. D. J. Thomas; demonstration of X rays.
Thursday, September 2: "Neurology", Dr. L. B. Cox; pathology demonstration, Dr. Bate.
Friday September 3: Ward round, Dr. M. L. Powell.
Tuesday, September 7: "Diagnosis and Treatment of Psychoneuroses", Dr. J. F. Williams; skin demonstration.
Thursday, September 9: "Recent Advances in Therapeutics", Dr. I. Maxwell; pathology demonstration, Dr. Rose.
Friday, September 10: Ward round, Dr. T. Frank.

On Tuesdays and Thursdays it is hoped to have patients available for examination from 1.30 p.m. to 2.15 p.m. A round will then be done of these patients from 2.15 p.m. to 3.30 p.m. From 4 p.m. to 4.50 p.m. there will be a lecture on the subject listed, and from 5 p.m. to 5.30 p.m. a demonstration of specimens, films *et cetera*. The Friday ward rounds will commence at 2.30 p.m.

The course will be limited to sixteen (16) students and a fee of £12 12s. will be charged. Applications for this course and inquiries should be addressed to Dr. J. Eric Clarke, c.o. the Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

The Royal Australasian College of Surgeons.

COURSE FOR FINAL F.R.A.C.S. EXAMINATION.

THE New South Wales State Committee of the Royal Australasian College of Surgeons is arranging a course of tutorial instruction in general surgery and pathology, commencing Monday, August 16, 1948.

This course will occupy sixteen weeks and will be administered by the New South Wales Post-Graduate Committee in Medicine in the University of Sydney.

The course is open only to candidates for the Final F.R.A.C.S. Examination in December.

Details may be obtained from the Secretary, New South Wales Post-Graduate Committee, 131, Macquarie Street, Sydney.

Obituary.

JOSEPH HENRY WILSON.

THE following appreciation of the late Dr. Joseph Henry Wilson has been received from Dr. J. McF. Rossell.

The death of Dr. J. H. Wilson at Orange on June 8 last at the age of eighty-five years severs a link with the pioneering days of medicine in western New South Wales. He practised in Warren from the early eighteen nineties to about 1902, and from then till he retired in 1927 in Orange and district. Since his retirement he had interested himself in grazing pursuits except for short periods as a locum for his friends.

As one who has known him intimately for some forty-five years as a family doctor and later as a partner and a friend, I feel, like many others of the west, that a great friend and adviser has gone. He was typical of the general practitioner and family doctor at his best and many went to him with their troubles, not all medical. All the many acts of kindness and generosity that he did were not known to any but the recipient in many cases. He used to "do good by stealth".

His great love of children made him popular wherever he went. His training at the Rotunda, his attention to detail, and his ability to improvise in things medical, helped to build for him a large midwifery practice. His clinical acumen and general professional ability kept him high in the opinions of his colleagues, and his moral and ethical standards caused him to be looked upon and sought as an adviser to medical men of the west.

His Irish outspokenness and his hatred of humbug were outstanding characteristics. He had a wide classical education which in those days was more general than now. He was a wide and liberal reader and his retentive mind made him an instructive and interesting conversationalist, especially on the earlier days of the west.

One son died as a prisoner of war in Germany in the recent war and this left a deep sadness over his later years. To the other children and to Mrs. Wilson the people of the Warren and the Orange districts extend their deepest sympathy.

GEORGE HENRY TAYLOR.

WE regret to announce the death of Dr. George Henry Taylor, which occurred on July 4, 1948, at Mosman, New South Wales.

COLIN CAMPBELL REID.

WE regret to announce the death of Dr. Colin Campbell Reid, which occurred on July 6, 1948, at Melbourne.

GEORGE CHRISTOPHER WALKER.

WE regret to announce the death of Dr. George Christopher Walker, which occurred on July 9, 1948, at Hobart.

FRANCIS EDWARD McAREE.

WE regret to announce the death of Dr. Francis Edward McAree, which occurred on July 11, 1948, at Melbourne.

THOMAS CONRAD REEVES.

WE regret to announce the death of Dr. Thomas Conrad Reeves, which occurred on July 12, 1948, at Melbourne.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Failes, John Watson, M.B., B.S., 1945 (Univ. Sydney), Box 98, Post Office, Coonabarabran.

Medical Appointments.

Dr. F. W. Arden has been appointed a member of the Queensland Health Education Council, in pursuance of the provisions of *The Health Acts*, 1937 *to* 1946, of Queensland.

Dr. F. J. Booth has been appointed a member of the Queensland Radium Institute, in pursuance of the provisions of *The Health Acts*, 1937 *to* 1946, of Queensland.

Diary for the Month.

JULY 27.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 28.—Victorian Branch, B.M.A.: Council Meeting.
JULY 29.—New South Wales Branch, B.M.A.: Branch Meeting.
AUG. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 4.—Victorian Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch **(Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.**

Victorian Branch **(Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.**

Queensland Branch **(Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.**

South Australian Branch **(Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.**

Western Australian Branch **(Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.**

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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